THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—45TH YEAR SYDNEY, SATURDAY, MAY 31, 1958 No. 22

Table of Contents.

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES— Page
A Survey of Shoe-Fitting X-Ray Fluoroscopes Located in New South Wales, by Alan Bell and H. M. Whaite .. 729
Sugar and Dental Caries, by C. Stanton Hicks .. 738
Hepatitis in "Q" Fever with Reports of Four Cases, by R. A. Douglas .. 739
Herpangina: Ulcerative Pharyngitis with the Isolation of Coxsackie Virus, by Ian Jack and R. A. Chenoweth .. 741
A Delayed Diagnosis of Bung Eye, by David J. Lee 743

REVIEWS—
The Peter T. Bohan Memorial Lectures on Medicine 743
The Century of the Surgeon .. 744
Studies on the Exo-Erythrocytic Cycle in the Genus Plasmodium .. 744
A Manual of Human Anatomy .. 744
Advances in Tuberculosis Research. Volume 8: B.C.G. .. 745
Headache: Diagnosis and Treatment .. 745
Clinical Electrocardiography: The Spatial Vector Approach .. 745
A Textbook of General Physiology .. 746
A Guide to Human Parasitology for Medical Practitioners .. 746

BOOKS RECEIVED .. 746

LEADING ARTICLES—
The Medical School of the University of Western Australia .. 747
The Menace of Shoe-Store Fluoroscopy .. 748

CURRENT COMMENT—
Entertainments in Hospital .. 748
Poliomyelitis .. 749
Medical Schools and Doctors of the World .. 749

ABSTRACTS FROM MEDICAL LITERATURE—
Medicine .. 750

ON THE PERIPHERY—
Why Not Motor? .. 752

CLINICO-PATHOLOGICAL CONFERENCES— Page
A Conference at Sydney Hospital .. 752

OUT OF THE PAST .. 756

SPECIAL CORRESPONDENCE—
London Letter .. 757

OBITUARY—
John O'Sullivan .. 758
George Hamlyn Harris .. 760
Mabel Irene Cousan Bray .. 760

CORRESPONDENCE—
Staffing of Base Hospitals .. 761
Anæsthesia in Heart Disease .. 761
National Health Service .. 761

POST-GRADUATE WORK—
The Post-Graduate Committee in Medicine in the University of Sydney .. 762
The Melbourne Medical Post-Graduate Committee .. 762

MEDICAL SOCIETIES—
Society for Clinical Science .. 762
The Endocrine Society of Australasia .. 762

NOTICE—
NAPT Commonwealth Chest Conference .. 763
British Medical Association, New South Wales Branch .. 763

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA .. 763

MEDICAL PRIZES—
The Darcy Cowan Prize .. 764
Australian Society of Anæsthetists' Prize for Anæsthetist Registrars .. 764

NOMINATIONS AND ELECTIONS .. 764

DEATHS .. 764

DIARY FOR THE MONTH .. 764

MEDICAL APPOINTMENTS: IMPORTANT NOTICE .. 764

EDITORIAL NOTICES .. 764

A SURVEY OF SHOE-FITTING X-RAY FLUOROSCOPES LOCATED IN NEW SOUTH WALES.

By ALAN BELL,
Director,

AND

H. M. WHAITE,
Senior Scientific Officer, Division of Industrial Hygiene, Department of Public Health, New South Wales.

All (ionising) radiations are potentially dangerous, their use should be the subject of constant and close scrutiny, . . . adequate justification should be required for their employment on however small a scale. There is a limit to the amount of radiation which any population or any individual can accept and we cannot afford to expend, without careful forethought, the margin which is now available to us (Medical Research Council, 1956).

In 1956 two important technical reports were published dealing with the many aspects of radiation; one document was prepared by the Medical Research Council and the other by the American National Academy of Sciences. In addition, numerous articles have also appeared in the English-speaking Press; in this respect Australian newspapers have been no exception. For these reasons, the general public is now considerably more "X-ray conscious". The New South Wales Division of Industrial Hygiene is currently carrying out surveys to determine the extent of radiation from some of the appurtenances of modern living.

The field work for our survey was carried out in Sydney during November, 1956, and in three country towns during June, 1957. In the metropolitan area, 14 stores were visited and 31 fluoroscopes tested; elsewhere units in six different shops were examined. Our objects were (i) to determine the extent of radiation received by customers and operators; (ii) to gain knowledge about X-ray techniques used in shoe shops.

DESCRIPTION AND USE OF A TYPICAL FLUOROSCOPE.

Such a machine, sometimes called a pedoscope, is inside a wooden box-like cabinet, about three and a half feet high, tapering from about one square yard at the base to about three feet by nine inches at the top. Along one side of the base, a nine-inch step leads to a raised platform about two square feet in area; at this level, the foot entrance is located.

Depending upon the make of machine, the X-ray tube may be in one of three positions, namely, below the foot and pointing upwards, above and pointing downwards or to one side of the foot. In the latter two cases, mirrors are used to reflect the image from the fluorescent screen.

In most instances there are three viewing ports at the top of the machine. The one near the operating

switch is used by the salesman; the central one is for the customer; the third, usually opposite the switch, is used by any other interested party such as the child's mother.

CAN ILL HEALTH BE CAUSED BY THE USE OF SHOE-FITTING FLUOROSCOPES?

It should be remembered that "most kinds of radiation damage are non-specific and any differential diagnosis involving radiation damage rests heavily on the availability of accurate estimates of the magnitude and type of exposure" (Albert, 1955).

Somatic Injury.

Although this type of fluoroscope has been used for many years, few instances of radiation damage have been recorded in the literature. One reason is the difficulty in establishing a clear-cut relationship; the long time interval between exposure(s) and the onset of disease makes such a task difficult. However, numerous histories of patients with skin and neoplastic changes occurring after years of latency amongst pioneers of diagnostic X radiation should suffice to remind us of the potential dangers.

In a recent issue of the *British Medical Journal* a case was reported of X-ray dermatitis with ulceration; for 10 years the sufferer had worked in a shoe store where a fluoroscope was used. The author of the article states: " . . . if, during transillumination, she [i.e. the victim] supported her right foot on the platform in front of the foot opening it was exposed to intense radiation . . . the site and development of the X-ray dermatitis showed it was mainly due to this" (Kopp, 1957).

The potential dangers were fully discussed by Hempelmann (1949) who stated that "several fittings within a day or two may conceivably lead to epiphyseal damage in children or to skin damage in children or adults. Repeated fluoroscopy of the foot by improperly regulated machines from early childhood to the age of normal closing of the epiphyses could result in foot deformities and even permanent skin damage".

Genetic Damage.

In recent months many authorities, both overseas and local, have criticized the use of these machines because of the now apparent need to avoid unnecessary radiation. We propose to quote a few opinions.

> The medical use of X-rays should be reduced as much as is consistent with medical necessity (National Research Council, 1956).
>
> It is folly to incur any X-ray exposure to the gonads which can be avoided (National Research Council, 1956).
>
> Long continued exposure to low intensity radiation induces as much gene mutation as a single high intensity radiation, provided that the total dose is the same. Experiments have shown that this probably remains true even when the dose is split into a series of small fractions, and no matter what interval elapses between the separate irradiations. Thus, in contrast to most other types of biological response to radiation, damage to genetic materials cannot be replaced and the effect from repeated exposures is cumulative . . . There is no known threshold for the induction of gene mutations by radiation; that is to say, any additional exposure, no matter how small, must be expected to raise the mutation rate, if only by a minute amount (Medical Research Council, 1956).
>
> If the whole population of the United States received a small dose of extra radiation, say one *r*,[1] then there is good reason to think that, among a hundred million children born to these exposed parents, there would be several thousand who would be definitely handicapped because of the mutant genes due to radiation . . . A population that is exposed, generation after generation, to an increased amount of radiation will experience a rising death rate and a falling birth rate . . . At present we are extremely uncertain about the level of this fatal threshold for a human population. This is one reason why we must be cautious about increasing the total amount to which the entire population is exposed (National Research Council, Washington, 1956).

[1] Rontgen (*r*) is a unit of X and γ rays. Millirontgen (*mr*) = $^1/_{1000}$ *r*.

LEGISLATION CONCERNING SHOE-FITTING X-RAY FLUOROSCOPES.

Numerous American States have regulations controlling the use of these units, e.g. Connecticut, Indiana, Mississippi, New Jersey, etc. In Victoria similar regulations were gazetted in 1951. We propose to review some of the salient features of both local and overseas legislation, so that later we may determine to what extent machines located in New South Wales conform to these requirements.

Victorian regulations require machines to be constructed in such a way that (i) the output shall not exceed 12 rontgens per minute as measured at the upper surface of the fluorescent screen (Schedule 3); (ii) the dose per exposure must not exceed one rontgen, and the exposure must then be terminated automatically (Schedule 3); (iii) the dosage rate in any region which may be occupied by the operator and/or any other attendant shall not exceed 12·5 millirontgens per hour (Schedule 6). In addition, a warning sign must be erected, stating that the customer must not view his feet more than three times in one day, nor more than 12 times per year. Likewise, feet should not be viewed unless shoes are worn. Other sections relate to the licensing of these machines, the position and type of switches, etc.

Similar standards for (i) and (ii) are required by the Division of Occupational Hygiene of the Massachusetts Department of Labour and Industry and by the Bureau of Industrial Hygiene of the Detroit Department of Public Health.

The Connecticut Sanitary Code Regulations limit the useful beam on the base of the foot opening to 0·7 *r* per minute in machines used for children, to 0·8 *r* in those used for women, and to 1·0 *r* for men. An automatic timer must be used to produce these dosages. In addition, stray radiation must not exceed 12·5 *mr* per hour at any of the following positions: (*a*) within six inches of the cabinet (except at the foot opening); (*b*) at the viewing ports; (*c*) 10 feet in front of the foot opening. These regulations also limit the total number of exposures to five per customer on any one day and to not more than 20 per year. The New York City Department of Health requires a five-second automatic cut-off, thereby limiting each exposure to two rontgens. Individuals must not "X ray" their feet more than 12 times a year.

Generally, the regulations of most American States limit the beam strength of machines used for male customers to 12 *r* per minute; about half the States specify lower limits for women and children, e.g. nine to 10 *r* per minute for the former, and six to eight *r* for the latter. Most codes specify a maximum dosage rate of 12·5 *mr* per hour in the vicinity of the operator.

STANDARDS ADOPTED BY US FOR THIS SURVEY.

When assessing the safety of foot fluoroscopes we adopted the following criteria: (i) The useful beam must not exceed 12 *r* per minute. (ii) The dosage rates at (*a*) the operator's position and (*b*) 10 feet in front of the foot opening must not exceed 12·5 *mr* per hour. (iii) The weekly permissible dose of radiation (*P*) for whole body exposure must not be more than 300 *mr* (Medical Research Council, 1956). (iv) Individual members of the occupationally exposed group (to X rays) should "not receive more than a total accumulated dose to the reproductive cells of 50 *r* up to the age of 30 years (by which age, on the average, over half of the children will have been born) and not more than 50 *r* additional up to the age of 40 years (by which time about nine-tenths of their children will have been born)" (National Research Council, 1956). These dosages are in addition to the radiation received from the natural background.

RADIATION MEASUREMENTS.

Techniques Used.

The physical measurements were made with a Victoreen *r*-meter, using a 25 *r* ionization chamber and an Ekco radiation monitor. The former instrument measured the

TABLE I.
Beam Intensities According to Position of Tube.

Tube Position.	Beam Intensity. (r per Minute.)		Number of Machines in Range. (r per Minute.)					Total Number of Machines.
	Average.	Range.	0 to 6.	6 to 12.	12 to 24.	24 to 48.	Over 48.	
Above	26·9	5·9 to 70·8	1	3	5	5	2	16
Below	14·9	2·2 to 51·8	4	5	8	1	1	19
Side	12·3	9·9 to 14·7	0	1	1	0	0	2
Total ..	20·0	2·2 to 70·8	5	9	14	6	3	37
Percentage ..	—	—	14	24	38	16	8	100

beam intensity in the foot opening, both directly and through a shoe, or shoes, selected at random. Three measurements were usually made and the results averaged.

The Ekco monitor, type N571, was used to assess the extent of scatter radiation from each machine at the following sites. (i) Three feet above the floor in the vertical line of the viewing openings used by the operator, customer and third person respectively (that is, in the approximate position of the gonads). (ii) At the corresponding viewing ports. (iii) At any nearby point where shop assistants might spend a considerable proportion of their time, e.g. at the cash register or counter. (iv) At the nearest seats for the customers. (v) Ten feet in front of the foot opening, both with and without a customer in position.

When considered necessary, salesmen were given film badges (Kodak Dental X-ray films type D.F. 11) to wear at breast level for one full working week. The exposed films were then sent to the Industrial Health Unit of the School of Public Health and Tropical Medicine, Sydney, for processing and dosage assessment.

Results Obtained.

Beam Intensities

Details may be found in Table I.

It will be observed that machines with overhead tubes had the highest beam intensities. The figures given are lower than those found during a 1950 American survey (Lewis and Caplan, 1950) when investigators calculated that the beam strengths of 40 machines averaged 38·4 *r* per minute; individual readings ranged from 12 to 107 *r* per minute.

In Table II beam intensities have been grouped according to the type of shoe department. It will be seen that machines used exclusively for boys, girls and younger

TABLE II.
Beam Intensities in Types of Shoe Departments.

Shoe Department.	Beam Intensity. (r per Minute.)		Number of Machines in Range. (r per Minute.)					Total Number of Machines.
	Average.	Range.	0 to 6.	6 to 12.	12 to 24.	24 to 48.	Over 48.	
Men's	23·6	2·2 to 70·8	2	1	3	2	1	9
Ladies'	23·2	7·8 to 34·4	0	3	4	0	2	9
Boys', girls' and children's ..	12·9	2·2 to 26·2	2	2	5	1	0	10
Ladies' and children's	28·2	11·3 to 39·6	0	1	1	2	0	4
All	15·2	5·9 to 34·8	1	2	1	1	0	5
Total ..	20·0	2·2 to 70·8	5	9	14	6	3	37
Percentage ..	—	—	14	24	38	16	8	100

TABLE III.
Dosage Rate According to Position of Tube.

Tube Position.	Dosage Rate to Operator. (*mr* per Hour.)		Number of Machines Giving Dosage Rate. (*mr* per Hour.)							Total Number of Machines.
	Average.	Range.	0 to 6·25.	6·25 to 12·5.	12·5 to 25.	25 to 50.	50 to 75.	75 to 150.	Over 150.	
Above	66	0 to 850	3	5	7	0	0	0	1	16
Below	216	1 to 1100	2	1	3	0	3	3	7	19
Side	136	2 to 270	1	0	0	0	0	0	1	2
Total	147	0 to 1100	6	6	10	0	3	3	9	37
Percentage ..	—	—	16	16	27	0	8	8	24	99

TABLE IV.
Dosage Rate at the Viewing Positions.

Viewing Position.	Number of Machines Giving Dosage Rate in Range (*mr* per Hour).							Total Number of Machines.
	0 to 6·25.	6·25 to 12·5.	12·5 to 25.	25 to 50.	50 to 75.	75 to 150.	Over 150.	
Operators'	6	6	10	0	3	3	9	37
Customers'	0	0	0	0	1	2	24	37
Third persons'	6	5	4	2	4	2	11	34

TABLE V.
Scatter Radiation Ten Feet in Front of the Machine.

Foot Opening.	Scatter Radiation Ten Feet in Front (*mr* per Hour).		Number of Machines in Range (*mr* per Hour).							Total Number of Machines.
	Average.	Range.	0 to 6·25.	6·25 to 12·5.	12·5 to 25.	25 to 50.	50 to 75.	75 to 150.	Over 150.	
No customer	32	1 to 100	8	3	8	11	2	5	0	37
Customer in place ..	16	0 to 65	12	7	11	6	1	0	0	37

children had generally lower beam intensities. In some of the major stores, this was a result of intentional adjustment.

Scatter Radiation.

In computing the figures for Tables III and IV, the higher dose rate (that it, the reading obtained at the position of the gonads or at the viewing port) was used.

Relationship to Tube Position.—It is apparent that the scatter radiation from machines with overhead tubes is approximately only one-third of that produced by those with the tube located below the customer's foot.

At the Three Viewing Positions.—From our observations, it appears that the third viewing position is rarely used. If it is, it is occupied either by the mother of the child being fitted or, on even rarer occasions, by another shop assistant acting as an interested viewer; in the latter event, scatter radiation would add little to the radiation dosage already being received by him in his official capacity.

Dosage rates at the customer's position are high because of the nearby port opening.

In the Vicinity of the Machine.—1. Ten feet in front. For this position measurements were paired, that is, taken when the machine was switched on and the foot opening alternatively unobstructed and when a customer had his foot in position. Averages show that in the latter circumstances the readings were approximately halved.

2. At nearby seats for waiting customers. Forty-seven locations were tested. In only two instances was the dosage rate in excess of 150 *mr* per hour.

Dosages Received.

When assessing whether or not a hazard exists, it is necessary to know the time for which the equipment operates, that is, the number and duration of exposures. In this respect we stress that operators' estimates were largely subjective and therefore probably liable to error. It is possible that the longer exposures did not last the full time stated, and vice versa for the shorter ones. However, we consider that the average of 10 seconds is fairly close to the true value.

By Customer per Exposure.—This value is obtained by multiplying the beam intensity in rontgens per minute (see Table I) by the duration of exposure. The results, therefore, are open to the errors discussed in the previous paragraph.

Approximately half the machines (54%) gave dosage per exposure of one rontgen or less; 73% delivered two rontgens or less. In the foregoing table it will be seen that the highest dosage per exposure was 86 *r*. Other investigators have reported a dose of 120 *r* received by a customer viewing a shoe fit (Spear, 1950).

By the Operator per Week.—In calculating these figures, we assumed that the salesman stood in the operating position only when he was pressing the switch; that he never bent over in front of the machine or stood behind a customer (e.g. holding a child's foot) while the unit was working.

In Table III it was shown that the dosage rate to the operators of foot fluoroscopes averaged 66 *mr* per hour with overhead tubes, and 216 *mr* when the tube was underneath, a ratio of 3·3. However, the actual dosage received

TABLE VI.
Times of Exposure.

Shoe Department.	Time of Exposure. (Seconds.)		Number of Machines with Exposure Time in Seconds.						Total Number of Machines.
	Average.	Range.	0 to 3.	3 to 5.	5 to 10.	10 to 20.	20 to 50.	50 to 100.	
Men's	8	2 to 20	2	4	0	3	—	—	9
Ladies'	22	2 to 100	5	2	0	0	0	2	9
Boys', girls' and children's ..	3·5	2 to 5	5	5	—	—	—	—	10
Ladies' and children's	3	2 to 4	3	1	—	—	—	—	4
All customers	10·5	3 to 40	3	1	0	0	1	—	5
Total	10	2 to 100	18	13	0	3	1	2	37
Percentage	—	—	49	35	0	8	3	5	100

TABLE VII.

Dosages Received by Customer per Exposure.

Shoe Department.	Dosage per Exposure. (Rontgens.)		Number of Machines with Dosage per Exposure of Rontgens.						Total Number of Machines.
	Average.	Range.	0 to 1.	1 to 2.	2 to 5.	5 to 10.	10 to 20.	Over 20.	
Men's	3·6	0·1 to 16·0	4	1	2	1	1	—	9
Ladies'	11·6	0·5 to 86·0	4	2	1	0	1	1	9
Boys', girls' and children's	0·8	0·1 to 2·2	8	1	1	—	—	—	10
Ladies' and children's	1·4	0·6 to 2·6	1	2	1	—	—	—	4
All customers	1·5	0·4 to 3·9	3	1	1	—	—	—	5
Total	4·3	0·1 to 86·0	20	7	6	1	2	1	37
Percentage	—	—	54	19	16	3	5	3	100

each week shows no such disparity. The average was 20 *mr* with the tube overhead, and 26 *mr* with the tube below, that is, a ratio of 1·3. This lower figure would indicate a generally shorter exposure time in the case of those machines with the tubes located below the customer's feet.

It was not possible to obtain more than 11 film badges from salesmen working in four stores (two in Sydney and two in country towns). The results are shown in Table IX.

In some instances, the radiation dose, as estimated by the film badge technique, exceeded the calculated figure; in others we found the reverse. This disparity was doubtless due to errors in estimating the time of exposure and number of exposures per week. The over-all average or weighted mean gave a weekly dosage of 25% of the permissible dose (*P*) to each operator; the corresponding mean for the film badges was 5% to 25% of *P*.

Conformity of the Machines to Our Standards.

1. Only 14 out of 37 machines (38%) had beam intensities less than 12 *r* per minute. The percentage of such machines in the boys', girls' and children's departments, namely 40%, was not significantly different from the over-all value.

2. In the operator's position, eight of the 16 "overhead-tube machines", and three of the 19 "underneath-tube machines", did not exceed 12·5 *mr* per hour.

3. Only 11 out of 37 machines (30%) conformed to the requirement of dosage rate not exceeding 12·5 *mr* per hour at a point 10 feet in front of the foot opening.

4. Only four machines (11% of the total) fulfilled all of the standards adopted by us for this survey, namely (i), ii (*a*) and ii(*b*).

5. The present international dosage standard (300 *mr*) was not exceeded, though it was closely approached by one machine.

6. "Genetic dosage." Under existing conditions it would be possible for sales people, working 50 weeks a year, for 12 years from the age of 18 to 30 years, to acquire a gonadal dosage greater than 50 *r* from some of these machines. At the worst two units, only three and 18 exposures per week would suffice. If time switches permitting a maximum of five seconds' exposure were fitted, these values would be increased to 55 and 71 exposures a week respectively; both of these units belonged to major city stores. The above numbers of screenings could easily be exceeded even when the general public was opposed to foot radiation.

TABLE IX.

Dosages Recorded by Film Badges.

Operator.	Calculated Weekly Dosage.		Recorded Weekly Dosage (Film Badge) as Percentage of *P*.
	Millirontgens.	Percentage of *P*.	
A	0	0	0 to 20
B, C, D, F	140	47	0 to 20
E	140	47	20 to 40
G, H	30	10	0 to 20
I, J	30	10	20 to 40
K	2	1	0 to 20
Average	75	25	5 to 25

The maximum weekly number of exposures claimed by any employee was 600. If the Victorian requirements of 12·5 *mr* per hour in the operator's position and maximum exposures of five seconds each were adhered to, the gonadal dosage to such an operator would be only 6·25 *r* over the 12-year period.

Table X gives details for all the machines seen.

Apart from the radiation received during the course of their duties, no fluoroscope operators had received sufficient other man-made radiation dosage to contribute

TABLE VIII.

Dosages Received by Operators per Week.

Tube Position.	Weekly Dosage (*mr*) to Operator.		Number of Machines with Weekly Dosage in Range (*mr*).						Total Number of Machines.
	Average.	Range.	0 to 7·5.	7·5 to 15.	15 to 30.	30 to 75.	75 to 150.	150 to 300.	
Above	20	0 to 290	14	0	1	0	0	1	16
Below	26	0 to 140	9	2	4	2	2	—	19
Side	8	0 to 16	1	0	1	—	—	—	2
Total	22	0 to 290	24	2	6	2	2	1	37
Percentage	—	—	65	5	16	5	5	3	99

TABLE X.
Dosage Received from Various Machines.

Exposure Time.	Number of Machines Giving Gonadal Dose of 50 *r* in 12 Years by Weekly Exposures.							Total.
	0 to 25.	25 to 50.	50 to 100.	100 to 250.	250 to 500.	500 to 1000.	Over 1000.	
As at present	2	0	1	3	4	0	27	37
If limited to five seconds	0	0	3	2	4	1	27	37

significantly towards the 50 and 100 rontgens permitted by the ages of 30 and 40 years, respectively. In Table XI, the cumulative dosages are set out for 16 employees, aged 40 years or less, who had experienced some exposure other than in the course of their work. In order to calculate this additional dosage, use was made of figures published by the Medical Research Council. When the dose of radiation received by the gonads in the course of X-ray diagnostic examinations etc., exceeded a few millirontgens, details are given in the column headed "Remarks". For calculating percentages, it has been assumed that the cumulative dosage (apart from the background) should be 0 rontgen at 18 years, increasing by just over four rontgens per year to a total of 50 at 30 years, then increasing at the rate of five rontgens per year to a total of 100 at 40 years of age.

Omitting consideration of the two girls both aged under 18 years and who, in our opinion, should not have been permitted to use these machines, it will be seen that in the 14 remaining cases, (*a*) the cumulative gonadal dosage from other sources did not reach 1% of the permitted dose, and in 10 of those cases it was negligible; and (*b*) with three exceptions, the cumulative gonadal dosage due to work was well under 5% of the permitted dose. In two of the three exceptions, the dosages were due to high scatter and a large total of exposures; in the other, to high scatter and long exposure times.

RESULTS OF QUESTIONNAIRE.

In order to gain information concerning day-to-day practices, either the person in charge of the department or the individual who operated the pedoscope most frequently was asked 34 questions; these concerned (i) X-ray techniques, (ii) habits of customers, and (iii) the machine and its environment. These subdivisions are not clear-cut, and it was not always possible to ask each salesman every question.

The following is a discussion of some of the most important conclusions.

Questions Relating to the Operator.

Who Operated the Machine?

In 24 out of 37 cases, any salesman in the shoe department was allowed to use the X-ray unit. For the remainder, usage was restricted to senior employees or supervisors. The ages of those concerned ranged from 16 to 73 years with an average age of 41 years. One salesman was not yet 16 years old, another was under 18 and four more were not yet 21 years old.

Training and Method of Using the Pedoscope.

When the salesmen were questioned about the training they had received before being allowed to use the machine, answers were often vague such as "very little" or "verbal only". For 16 of the units examined (43%), the matter of training had not been considered sufficiently important by the management to be given only by senior staff. Five salesmen said that they had received no instruction whatsoever.

TABLE XII.
Operator's Guide in Fitting.

Guide in Fitting.	Number of Operators.	Percentage.
Bone shadow	28	78
Soft tissue outline ..	3	8
Both	4	11
No idea	1	3
Total	36	100

Operators were asked "What guides you in the fitting—the bone shadow or the outline of soft tissue?" The replies received are given in Table XII.

During the initial part of our survey, that is before there had been any of the recent adverse Press publicity on X rays, many assistants replied a definite "yes" when asked if the machine was a necessity and of real use to them. Some months later, when some of these employees were questioned again, we found that they had changed their minds and, as a result, machine usage had either substantially dropped or had been discontinued. For example, in one store shop assistants had refused to use the unit;

TABLE XI.
Estimated Cumulative Gonadal Dosage Received by Sixteen Operators.

Operators' Ages in Years.	Operators' Sex.	Estimated Permissible Cumulative Gonadal Dose (*r*).	Cumulative Gonadal Dosage. (*mr.*)			Cumulative Dosage as Percentage of Permissible Dose.			Previous Exposures.
			Work.	Other.	Total.	Work.	Other.	Total.	
40	M.	100	550	130	680	0·6	0·1	0·7	X-ray of spine.
40	F.	100	2200	50	2250	2·2	0·0	2·2	Fitting own shoes.
26	F.	38	400	1	400	1·2	0·0	1·2	
40	F.	100	550	1	550	0·6	0·0	0·6	
38	F.	90	120,000	1	120,000	133·0	0·0	133·0	
32	F.	60	21,000	2	21,000	35·0	0·0	35·0	
30	M.	50	600	1	600	1·2	0·0	1·2	
33	M.	65	750	40	790	1·1	0·1	1·2	Fitting own shoes.
40	M.	100	550	710	1260	0·6	0·7	1·3	X-ray of femur.
29	M.	46	22,000	1	22,000	48·0	0·0	48·0	
20	F.	8	250	1	250	3·1	0·0	3·1	
35	F.	75	425	600	1025	0·6	0·8	1·4	Three abdominal X-rays.
16	F.	0	25	1	26	—	—	—	
16	F.	0	25	1	26	—	—	—	
27	F.	38	225	1	226	0·6	0·0	0·6	
40	M.	100	550	1	550	0·6	0·0	0·6	

in another, the switch had been deliberately put out of action by employees, and the management had not bothered to have it repaired; in other places the pedoscopes had been taken away from the shoe department. Some other stores had adopted a policy of actively discouraging the public, and stated that they told inquiring customers that the machine was in need of repairs and, therefore, could not be used.

Knowledge of X Rays and their Effects.

Twenty-two out of 38 employees stated that they had no knowledge whatsoever about the properties of X rays; 12 said they understood the subject reasonably well and realized that there were certain hazards; one of these persons was a nurse.

The technical knowledge of some supervisors, who instructed new salesmen, was extremely poor. For example, some did not know that the rays were invisible; others failed to realize that it was potentially dangerous to stand in front of the foot opening while the machine was switched on.

Five women, all working in departments handling children's shoes, said that they often held the child's foot in position while somebody else operated the machine. The need for this arose only rarely with adults, e.g., with frail and unsteady elderly women. Protective gloves were not worn. When employees were asked if they demonstrated the use of the machine with their hands or feet, seven replied "yes, frequently", four said "occasionally", two "rarely" and 22 denied doing so.

Previous Film Badge Tests.

Only three operators had previously worn film badges; two of these people worked in country shops where the machines were used considerably less frequently than in some of the major city stores.

Questions Relating to the Customer.

Number of Previous Exposures.

Thirty-seven out of 38 salesmen, said that they did not ask the customer how many previous foot X rays he or she had had. A frequent reply to our question was, "It would be bad sales psychology to make such inquiries as it would make the machine out to be dangerous".

Is the Customer Warned of any Limit to the Number of X Rays?

Table XIII shows the replies received.

Only at one store was there a suitable warning notice. This particular shop was a large one and had had another somewhat similar placard in their children's department. This second notice had been mislaid some time prior to our visit and unfortunately had not been replaced.

Number of Viewings.

In numerous stores, the salesmen agreed that in many instances it was very difficult, if not impossible, to prevent adamant customers from having multiple screenings. However, from our inquiries, it appeared that, apart from a small minority, repeated viewing at the same shop was not a common practice.

Whether or not buyers viewed both feet simultaneously did not necessarily depend upon the size of the foot opening. To our questions eight salesmen said that "most customers viewed one foot at a time"; 22 replied "both feet simultaneously" and eight replied "sometimes one foot, sometimes two". In all probability, the time taken to view two feet together is less than the total time spent looking at them separately.

From discussions with salesmen it would seem that, at least until a short time ago, a large percentage of the people buying shoes liked and expected to have their feet X rayed. However, as previously stated, due to Press statements the pendulum has now swung in the opposite direction; it should not be forgotten that on such topics the public's memory may be short. We were frequently told that prior to this publicity, 75% to 95% of children's fittings were made with the help of pedoscopes. In many instances, customers would walk out of the store without buying if such a unit was not present.

Reasons for Using the Machine.

Thirty-seven out of 43 salesmen stated that they used the machine in the final stages of fitting; in most instances the shoes had already been more or less decided upon. Only occasionally was the machine used to persuade the customer that he had made a bad choice. Sometimes mothers would bring children along with old shoes and expect to use the machine to determine if the shoes were ready to be discarded or not.

Of 25 assistants interviewed, 20% stated that in the case of cripples, etc. the machine was a great help to them.

TABLE XIII.
Warnings Given to Customers.

Type of Warning.	Number of Persons Questioned.
No warning	24
Occasional verbal warning	6
Regular verbal warning	1
Reliance on warning sign	1
Limit of two to three exposures	4
Limit of 20 seconds' to two minutes' exposure	2
Total	38

Viewing of the Unprotected Foot.

Eleven salesmen, using nine machines, said that they had seen customers place their foot in the machine, for viewing, when not wearing shoes. The likelihood of this undesirable practice happening is greatly increased if the machine switch is in such a position that it can be operated unbeknown to the operator.

Transmission Measurements.

Six of the nine units mentioned in the preceding paragraph had their tubes mounted above the foot; for the other three they were located below. In the former case, because only the thin leather upper of the shoe intervened, there was little reduction in beam intensity.

TABLE XIV.
Transmission Measurements for Machines with Overhead Tubes.

Type of Shoe and Upper.	Transmission.
Men's shoes, leather uppers	0·76
Women's shoes, leather uppers	0·74
Boys' and girls' shoes, leather uppers	0·80
Children's shoes, leather uppers	0·83
Children's sandals, open leather uppers	0·90

Transmission measurements were made on a total of 17 shoes in the beams of the 16 machines with overhead tubes. Table XIV shows the average figures obtained.

It will be seen, therefore, that with overhead tubes the reduction in beam intensity ranged between 10% and 26%.

Where the tube is mounted below the foot there is an appreciably greater reduction in beam intensity (between 43% and 80%) due to the interception of the sole of the shoe. On 19 units of this type transmissions were measured for a total of 26 shoes (Table XV).

From a consideration of the original figures, it can be shown that placing the unshod foot in the direct beam would result in an increase in dosage of about 10% to 35% for overhead tubes, and of about 75% to 1300% for tubes underneath, compared with what is normally experienced wearing a shoe.

Questions Relating to the Machine and its Environment.

Switches.

Only five machines had an incorporated tumbler switch, which is designed to prevent unauthorized use of the plunger. In a few cases, the more tedious practice of disconnecting at the power-point or switching off at a remote master switch was used for that purpose.

There were no true time switches limiting exposures to a matter of seconds. One firm had three machines equipped with a timer, which limited the exposure or a series of successive shorter exposures to between one and one-and-a-half minutes from the initial pressing of the plunger. Since the beam strengths of these three machines were 4·1, 9·5 and 13·0 rontgens per minute, dosages to a customer could have ranged from 4·1 *r* with a one-minute exposure, to 19·5 *r* with a one-and-a-half minute exposure. Clearly these devices made no real contribution to the limitation of foot dosage.

No machines had separate switches for men, women and children. The purpose of such switches is to produce beams of decreased intensity for X raying the feet of the last two classes of customers.

TABLE XV.

Transmission Measurements for Machines with Underneath Tubes.

Type of Shoe and Sole.	Transmission.
Men's shoes, leather soles	0·41
Men's shoes, rubber soles	0·07
Women's "heavy" shoes, leather soles	0·50
Women's fashion shoes, leather soles	0·46
Boys' and girls' shoes, leather soles	0·26
Boys' and girls' shoes, "Wearite" soles	0·34
Girls' party shoes, leather soles	0·55
Children's shoes, leather soles	0·39
Children's shoes, "Kromhyd" soles	0·57
Children's shoes, crepe soles	0·51
Children's shoes, "Wearite" soles	0·20

Warning Notices.

Only seven of the 37 machines had a nearby notice informing the public that the machine should be operated only by authorized personnel. Some assistants admitted that they had, on occasion, "left the customer having a look either while attending to other buyers or while answering the phone". In such circumstances, the exposure time could be very long.

Location of Machine.

Nineteen machines were placed against pillars. Apart from being a suitable position for the machine, a pillar provides a convenient location for a power point. Some other locations may give concern to the casual observer, e.g., when the machine is against the side of a flight of stairs or beside a serving counter. However, in the first of these cases, though the steps were continuously used by shoppers, they were above the level of the machine, and therefore safe; in the latter case, a dosage rate of only 0·2 *mr* per hour could be detected behind the counter.

In general, shop assistants move around and are unlikely to be in front of the foot opening when the machine is working. However, customers may be seated in chairs directly in front of the foot opening and, in busy periods, may be waiting some time before being served. During this survey, observations were made concerning the distance and direction of the nearest (and therefore most exposed) seats relative to the foot opening. Seat positions are summarized in Table XVI.

In two of these seats, both directly in front of the opening, one at five feet and the other at six feet, the maximum reading of 150 *mr* per hour on the Ekco radiation monitor was exceeded. From observations made at greater distances, the dosage rates at these seats were about 220 *mr* per hour in both cases. A customer sitting in one of these seats on a busy day might conceivably be exposed to scatter radiation from, say, 12 exposures of five seconds each. The dosage to that customer would be between three and four millirontgens, which is little more than 1% of the permitted weekly dosage of 300. However, even this low amount is unnecessary, and it could be reduced by better positioning of the machine and seats.

In one store, a child's rocking horse was situated within three feet of the machine. This toy was used by youngsters while their mothers shopped. Although stray radiation at this site was less than one millirontgen per hour (the firm did not know this), we consider that this type of practice is unsound.

TABLE XVI.

Position of Most Exposed Seats Relative to Foot Opening.

Position of Seats.	Number of Machines.	Percentage.
Directly in front (5° each side of centre line):		
(*a*) Up to five feet away	2	5
(*b*) Over five feet up to 10 feet away	9	24
(*c*) Over 10 feet away	3	8
Partly to side (5° to 30° each side of centre line):		
(*a*) Up to five feet away	1	3
(*b*) Over five feet up to 10 feet away	5	14
(*c*) Over 10 feet away	1	3
Well to side (30° to 90° each side of centre line):		
(*a*) Up to five feet away	4	11
(*b*) Over five feet up to 10 feet away	9	24
(*c*) Over 10 feet away	2	5
Behind the machine	1	3
Total	37	100

Ease of Viewing.

In no case was the section of the department darkened where the machine was installed. In three instances there was interference with the viewing, mainly because of glare from the afternoon sun, a skylight or nearby neon lights. In almost all cases the operator made little or no attempt to accustom his eyes to the darkness before switching on the machine.

In our opinion the screen brightness of a number of the machines was very dull and we considered that it was difficult to obtain a satisfactory viewing image.

TABLE XVII.

Possible Reductions in the Size of Viewing Ports.

Size of Entrance Port. (Square Inches.)	Average Area. (Square Inches.)	Number of Machines.	Number of Machines where Reduction Favoured.	Percentage of Operators Favouring Reduction.	Average Reduced Area Favoured. (Square Inches.)
50 to 80	67	24	11	46	48
100 to 140	128	13	8	62	66
Total	88	37	19	51	56

Age, Maintenance and Testing of the Machine.

It was not always possible to ascertain how long the machine had been in use. However, 30 out of 35 machines were of post-war origin; one unit had been in use more than 20 years. Fifteen of the city machines examined had been checked instrumentally for scatter radiation in 1951 or 1952 by the Director of the State Bureau of Physical Services. In the country, two units had been tested by radiologists using film badges.

Foot Entrance Ports.

None of these openings were curtained with leaded rubber or other radiation-absorbing material. Shop

assistants were asked whether or not, in the light of their experience, the size of these openings could be reduced. Their replies are summarized in Table XVII.

CONCLUSION.

From our survey it will be apparent that the existing usage of foot X-ray machines, whether viewed from the point of view of radiation exposures to the customer and salesman or from the point of view of existing operating practices, leaves much to be desired in New South Wales.

In many instances, the measures necessary to bring offending machines within the physical standards discussed are simple. For example, the beam strength could be reduced by lowering the tube voltage and/or current. A further reduction in dosage rate, without appreciably affecting the screen brightness, could be achieved by increasing the effective filtration to the equivalent of three millimetres of aluminium. Figures given in the recommendations of the International Commission of Radiological Protection show that this reduction is more than 50% when, at 60 kilovolts (pulsating) and three milliamperes, the total filtration (aluminium equivalent) is increased from one and a half to three millimetres. Scatter radiation from the foot opening could be reduced by the placing of strips of leaded rubber in this position; this is done on some English machines (Dyson, 1956).

We consider that the likelihood of somatic damage resulting from the machines we examined is very slight. However, we agree with the current opinion, which is now almost world-wide, that from the genetic point of view it is undesirable to expose individuals, whether customers or salesmen, to unnecessary radiation. Our survey showed that some employees could receive during their working life, and in fact one already had received, a gonadal dose greater than that recommended.

Many authorities in various countries have expressed the view that such machines should not be used in shoe shops. For the readers' convenience we propose to quote a few opinions.

D. J. Stevens, Director of the Commonwealth X-ray and Radium Laboratory, Melbourne, stated that "it is important that persons should preserve their ration of permissible radiation, and not dispose of it on non-essentials such as footwear fluoroscopes" (statement made at the Forty-fourth Session of the Industrial Hygiene Committee of the National Health and Medical Research Council of Australia).

"There seems little if any excuse for X-ray examination of the feet of customers in shoeshops . . . it adds unnecessarily . . . to the difficulty of keeping within the maximum permissible dose . . . It should be abolished" (MEDICAL JOURNAL OF AUSTRALIA, March 30, 1957).

E. P. George, Director of Medical Physics, St. Vincent's Hospital, Sydney, stated that "I'd gladly see them abolished . . . of all X-ray appliances the pedoscope is by far the most dangerous . . . the machine is a useless and dangerous sales gimmick" (*Daily Telegraph*, May 13, 1957).

"The practice of routinely examining the feet by X-rays when fitting shoes is of dubious value . . . it is in our view hardly justifiable. We hope that the procedure will be abandoned, except when prescribed for orthopædic purposes" (Medical Research Council, 1956).

The previous extracts are also our own personal opinions; we stress that our views are not necessarily those of the New South Wales Department of Public Health. We understand, from personal correspondence, that some State Radiological Advisory Councils will recommend the banning of such units. From our inquiries, it would appear that if licences for pedoscopes were refused in New South Wales, those stores concerned would raise little or no objection provided all shoe stores were affected equally.

It is interesting to note that whereas many people have stated that such machines are not necessary, it would appear that their efficiency and usefulness have never been thoroughly investigated. It would be interesting to hear the views of an authoritative body perhaps composed of a radiologist, an orthopædist, a physicist and representatives from the suppliers and users.

As previously stated, we agree with others that the use of pedoscopes should not be allowed in shoe shops. Should this be impracticable for any reason, we consider that the next best course of action would be to restrict their usage to "licensed" personnel. Such a person should have (*a*) the necessary status and authority to be able to resist unnecessary customer demands; (*b*) adequate technical knowledge so as to be able to use the machine safely; (*c*) medical knowledge about foot abnormalities and diseases. This type of licensing system has recently been introduced in parts of America; e.g., in New York State, in May, 1957, the Health Commissioner approved an amendment to the Sanitary Code barring the operation of the machines producing radiation by anyone other than properly qualified persons.

In order to train salesmen to meet our suggested requirements it would be necessary to conduct a special course; this would cost money and, on completion of the course, the personnel would probably command salaries greater than those currently paid to existing salesmen. Many stores might not find it economically possible to employ such a person. Finally we would like to point out that in the latter scheme, first, it would still be impossible to ensure that customers were not having more than the recommended number of exposures per year. Secondly, licensed pedoscopes would have to be inspected and tested at regular intervals by trained State officers. This would involve the use of valuable man-hours which could be more profitably spent on some of the major and important applications of radiation.

ACKNOWLEDGEMENTS.

This survey would not have been possible but for the willing cooperation and friendly advice of numerous people. In particular we wish to express our thanks to the management and staff of the numerous stores and shops visited; to Mr. B. W. Scott, Director of State Bureau of Physical Services, and to Dr. S. F. Marshall, of the Division of Industrial Hygiene. Finally, we wish to thank the Director-General of Public Health for permission to publish this article.

REFERENCES.

ALBERT, R. E. (1955), "Criteria for the Diagnosis of Occupational Illness Radiation", *Indust. Med.*, 24 : 436.

DEPARTMENT OF HEALTH, VICTORIA, AUSTRALIA (1951), "Regulations Relating to the Use of X-Ray Fluoroscopy in Shoe Fitting", September.

DYSON, E. D. (1956), "Shoe Fitting X-Ray Fluoroscopes: Radiation Measurements and Hazards", *Brit. M. J.*, 2 : 269.

HEMPELMANN, L. H. (1949), "Potential Dangers in the Uncontrolled Use of Shoe Fitting Fluoroscopes", *New England J. Med.*, 241 : 335.

KOPP, H. (1957), "Radiation Damage Caused by Shoe Fitting Fluoroscope", *Brit. M. J.*, 2 : 1344.

LEWIS, L., and CAPLAN, P. E. (1950), "The Shoe Fitting Fluoroscope as a Radiation Hazard", *California Med.*, 72 : 26.

MEDICAL RESEARCH COUNCIL (1956), "The Hazards to Man of Nuclear and Allied Radiations", Cmd. 9780, H.M. Stationery Office: 16, 29, 67, 69, 87, 116.

NATIONAL RESEARCH COUNCIL (1956), "The Biological Effects of Atomic Radiation", A Report to the Public National Academy of Sciences, Washington: 8.

NATIONAL RESEARCH COUNCIL (1956), "The Biological Effects of Atomic Radiation", Summary of Reports to the National Academy of Sciences, Washington: 18, 29, 30.

New York Times, June 1, 1957 : 1.

SPEAR, F. G. (1952), "The Biological Response to Penetrating Radiations", in "Biological Hazards of Atomic Energy", being papers read at the conference convened by the Institute of Biology and the Atomic Scientists Association, October, 1950, Oxford Clarendon Press: 2.

UNITED STATES DEPARTMENT OF HEALTH, EDUCATION AND WELFARE, PUBLIC HEALTH SERVICE (1954), "Occupational Health and Safety Legislation", compilation of State Laws and Regulations, Publication No. 357, U.S. Government Printing Office, Washington.

SUGAR AND DENTAL CARIES.

By C. STANTON HICKS,
Professor of Physiology, University of Adelaide, and Scientific Adviser on Foodstuffs and Feeding to the Australian Military Forces.

WHILST being conducted over an excellent seaside summer camp for country children of both sexes, I was struck by a demonstration set-up organized by the Victorian Association for Dental Health on the causal relation between the dietary intake of sugar and the incidence of dental caries. The propaganda, as such, was really good, but is the basis for the propaganda scientifically sound? In all such laudable efforts to improve health by education, it seems a pity to create a possible adverse reaction by using statements that are subject to criticism, and which may, in the future, undermine such efforts, however well they are intended.

The cause of caries is still obscure. It is being made more obscure by dogmatic statements, some of which are arousing world-wide discussion and even strong opposition on both scientific and social grounds. If it is granted that the cause of caries is obscure, it is desirable to examine the basis for indoctrination of children on the contribution to the ætiology of caries made by sugar or by carbohydrates generally.

The inculpation of sugar dates from 1890 when the S. S. White Dental Manufacturing Co., of Philadelphia, published a report by W. D. Miller on the possible connexion between bacterial fermentation of starch in the mouth and the production of acid and the consequent solution of the mineral structure of the teeth. From this process of hydrolysis of large carbohydrate molecules to sugars, the next step was to conclude that the ingestion of pure sugar was even more likely to lead to caries. H. P. Pickerill was a strong protagonist of the mastication of acid fruit such as apples prior to retiring at night, on the ground that this evoked an alkaline salivary flow which inhibited the above-mentioned acid formation.

Mellanby (1923) introduced the notion that tooth structure was an important underlying factor in the incidence of caries, and this in turn led to consideration of metabolic aspects of the ætiology (Mellanby, 1928). Then followed the discovery that fluoride as a metabolic factor might affect either the structure and composition of the teeth, or the oral environment.

This brief conspectus brings us to 1953. The present communication refers specifically to the extensive and rigorous study of caries conducted at the behest of the Swedish Government by the Royal Medical Board of Sweden at the Vipeholm Hospital, Lund, and to Special Report Number 288 of the Medical Research Council of Great Britain. Both of these reports deal with the influence of carbohydrates, including pure sugar, upon the incidence of caries, and bring the results of authoritative research up to the end of 1955.

The Vipeholm studies were undertaken because the Swedish Government became alarmed at the high incidence of caries in the teeth of recruits for the armed services. The report therefore summarizes the literature up to the start of the investigations, as a guide for the authorities. The first conclusion drawn from this examination is that: "It is not easy to decide whether or not caries activity in man is related to the carbohydrate intake." Further, it is concluded that:

> 1. Caries activity is as a rule low in institutionalized children. No definite conclusion can be made concerning the cause of this low level.
>
> 2. The effect of increased sugar ration on dental caries seems not to be entirely due to the amount of sugar but also to other factors.
>
> 3. Reduction in dietary sugar is followed by a reduction in activity of dental caries but not to the extent often supposed.

Finally, this portion of the report concludes that:

> The discrepancy of the results obtained in these studies also explains the sceptical attitude assumed by many objective workers in this field towards the value of encouraging large scale reduction of carbohydrate intake in anti-caries campaigns.

The foregoing is the summary of a critical examination of the investigations on the subject carried out by 65 research workers mostly since the war.

The Vipeholm studies were carried out on 436 individuals for a period of five years. The questions posed were: "Does an increase in carbohydrate (sugar) intake cause an increase in dental caries? If so, what factors related to consumption are of importance? Does a decrease in carbohydrate (sugar) intake produce a decrease in dental caries activity?"

The planning, the techniques used and the recording of results have been the subject of the greatest critical examination, and as the report is available in English, there is no need to do more than cite the results, which are as follows:

> 1. The consumption of sugar increases caries activity.
>
> 2. The risk of sugar increasing caries activity is great if the sugar is consumed in a form with a strong tendency to be retained on the surfaces of the teeth.
>
> 3. The risk of sugar increasing caries activity is greatest if the sugar is consumed between meals and in a form in which the tendency to be retained on the surfaces of the teeth is pronounced with a transiently high concentration of sugar on these surfaces.
>
> 4. The increase in caries activity under uniform experimental conditions varies widely from one person to another.
>
> 5. Increase in caries activity due to intake of sugar rich foodstuffs consumed in a manner favouring caries disappears on withdrawal of such foodstuffs from the diet.
>
> 6. Caries lesions may continue to appear despite the avoidance of refined sugar, maximum restriction of natural sugars and total dietary carbohydrates.

Here, as a result of a peculiarly critical study, we find, for the first time, direct evidence of a relation between the caries process and sugar consumption—but, let it be noted, consumption of sugar in a particular sticky form. The commission found that: "When sugar was consumed in solution at meals up to more than twice the Swedish consumption, no increase in dental caries was observed."

The report concludes with a searching statistical analysis of the results. These deserve quotation, and are as follows:

> 1. The method for determining caries activity had inherent systematic errors. There was a slight difference between the criteria of the two examiners concerning what should be regarded as caries in initial stages in fissure systems and on facial surfaces.
>
> 2. The deviations seemed to consist mainly of a tendency to under-registration.
>
> 3. The differences were of the same order for both examiners and appeared to be independent of the caries activity of the material.
>
> 4. In a series with low mean activity (1-3 new carious surfaces per person per year) the error of a single observation was of the order of 1 new carious surface. A mean of caries activity based on 40-50 individuals thus has an unreliability of on the average 0·2 new carious surfaces per person per year.

The Medical Research Council Report is published over the signatures of King, Mellanby, Stones and Green. In outline, the study comprised the following:

> 1. Observations of the incidence and spread of dental caries in groups of children in residential homes over a period of one year on their ordinary diet, in order to ascertain their "natural" caries increase (1948 to 1949).
>
> 2. The addition of sugar, in the forms in which it is normally consumed, to the diets of some of the children for periods of one, one-and-a-half, and two years, and a comparison of their dental condition at the end of these periods with that of control children on unsupplemented diets (beginning 1949).

Again there follows a critical examination of the methods used and the results of studies carried out in three separate centres—in London, Liverpool and Sheffield. The authors state that at the outset of the work scientific proof of the implication of sugar intake on caries incidence was lacking.

To quote the report:

> The results of the investigation are, in the main, clear cut and therefore can be stated quite briefly. They demonstrate that relatively great differences in the total sugar content of the diets of children in institutions had no significant effect on the initiation or spread of dental caries in periods of one to two years.

Finally:

> Thus, the results of the three separate studies, which were geographically quite distinct and were conducted by three observers who knew nothing of each other's results until the end, are all essentially similar. It seems probable that the apparent anomalies in the findings for both deciduous and permanent teeth are chance variations due to small numbers when the main groups of children are split into sub-groups, since they show no consistency when the results are surveyed as a whole; it also seems unlikely that sugar affects the two dentitions differently.

This report should be read in conjunction with the Vipeholm Report because the latter contains evidence gained from studies of the effect of sticky sweets—the only direct evidence of a causal connexion between caries and sugar intake. On the other hand, the Medical Research Council Report makes the following statement at the conclusion:

> While this investigation does not prove that the sugar content of the diet, *however distributed and however eaten*, never affects the teeth, it certainly points to the practical conclusion that, provided a child is fed on a diet as good as that of the average children's residential institution in England, a substantial increase in the amount of sugar in the diet will not affect the child's liability to dental caries within a period of at least two years.

These two major studies of the relation between sugar intake and the incidence of caries or caries activity would appear to merit wider recognition. From the viewpoint of one actively engaged in the field of practical nutrition on the large scale, it is reassuring to learn that, so far as these researches go, sugar intake is a real factor in caries activity only when it is consumed in a form which ensures that it sticks to and acts in a concentrated form upon the surface of the tooth. The fact that it is most effective when it is consumed between meals, only emphasizes the complex nature of the effect of sugar in dental caries. Sweetened drinks, on the other hand, can be consumed with impunity so far as sugar content is concerned. This is a matter of considerable importance in the feeding of troops in the tropics..

The Effect of Acid Drinks.

Although this communication merely reports the latest authoritative work on sugar in relation to the carious process, this reference on the effect of acid drinks is added in response to a request. A review of the most recent work indicates that the erosive action of acids, as well as of sugar, takes place after close contact, and it depends on the duration of contact. Sucking lemons and oranges can destroy the teeth, but citrus drinks do not. This is well known to the medical profession; when hydrochloric acid is continually prescribed, it must not be taken through or over the teeth, but interspersed with food. The original work of McKay implicating acid drinks in tooth erosion was done on rats drinking from a glass tube direct onto the teeth. He has since reexamined these results and modified his opinion (McKay *et alii*, 1945). The work of Nuckolls *et alii* (1952) disposes quite conclusively of the concept of the erosive action of acidulated drinks. The acids studied in commercial juices, cordials and aerated drinks were citric, tartaric and malic; phosphoric, acetic (in pickles) and lactic (in butter milk) were also studied.

References.

BONTING, S. L. (1952), "The Effect of a Prolonged Intake of Phosphoric Acid and Citric Acids in Rats", *Joh. Erschede en Zonen*, Haarlem.

HÖJER, J. A., and MAUNSBACH, A. B. (1954), "The Vipeholm Dental Caries Study", *Acta odont. scandinav.*, 11:195.

KING, J. D., MELLANBY, M., STONES, H. H., and GREEN, H. N. (1955), "The Effect of Sugar Supplements on Dental Caries in Children", Medical Research Council of the Privy Council, Special Report Series No. 288, H.M. Stationery Office, London.

MCKAY, C. M., RESTARSKI, J. S., and GORTNER, R. A. (1945). "The Effect of Acid Beverages Containing Fluoride upon Teeth of Rats and Puppies", *J. Am. Dent. A.*, 32:668.

NUCKOLLS, J., HALDI, J., BLAKE, W. R., and MUNSON, C. S. (1952), "Submissions to Select Committee on the Use of Chemicals in Food Products", Proceedings of the House of Representatives, No. 74.

HEPATITIS IN "Q" FEVER WITH REPORTS OF FOUR CASES.

By R. A. DOUGLAS,

Visiting Physician, Townsville General Hospital, Townsville, Queensland.

THE occurrence of hepatitis in "Q" fever, as an important manifestation, has received scanty attention in the literature on the disease, despite the fact that in Derrick's (1937) original description of the disease one of his patients had hepatitis. "Only one patient in this series became jaundiced. This was number VII, the most severely affected of them all. The jaundice appeared on the thirteenth day of the illness, deepened during the next week and disappeared in another ten days." In their series of 180 cases of "Q" fever in California, Clark *et alii* (1951) noted that 5% of patients were jaundiced. Hepatomegaly and liver tenderness were observed in 11% and 7% of the patients respectively. "Until it was recognised that such evidence of hepatic involvement was consistent with the diagnosis of 'Q' fever, these cases gave rise to considerable difficulty in the clinical differentiation from infectious hepatitis. Ordinarily, evidence of hepatic involvement became apparent in the second or third week of illness, and most of the patients with such evidence had a prolonged clinical course and a slow convalescence." Ludwig (1956) described two cases of "Q" fever hepatitis and reviewed the world literature. At that time he was able to find clinical reports of a total of only 15 jaundiced patients. His two Swiss cases made a total of 17 reported cases. In one of his cases jaundice appeared on the seventh day of the illness and lasted for eight weeks, and in the other case it appeared on the fifth day and lasted for two and a half weeks. He was doubtful whether or not he was dealing with a fortuitous coincidence of the two diseases, infective hepatitis and "Q" fever, but finally decided, for various reasons, that the cases must have been true cases of "Q" fever hepatitis. Wespi (1957) added a further reported case of a patient with initial typical pneumonia who later developed hepatitis.

Textbooks of medicine are largely silent on the problem, and this may be responsible for the average clinician's ignorance of the frequency of hepatitis in "Q" fever. Thus Megaw (1952), Snyder (1955) and Sherlock (1955) do not mention the occurrence of hepatitis in "Q" fever. Maegraith (1956) does state: "In those cases in which the febrile episode continues for weeks, however, signs of liver involvement, including jaundice, may develop." In none of the foregoing textbooks is any mention made in the accounts of infective hepatitis that "Q" fever should be considered in the differential diagnosis.

Reports of Cases.

CASE I.—The patient was a male, aged 50 years. He had worked for two years in the Townsville area as a railway fettler. There was no recent known contact with cattle or other animals. He was admitted to the Townsville General Hospital on July 2, 1956, with a history of anorexia, nausea, malaise and upper lumbar pain for one week. The urine had been dark for two days. Physical examination

showed him to be a slightly icteric man with a tender, enlarged liver palpable two fingers' breadth below the right costal margin. No other abnormality was detected. His temperature was 100° F., and he had a slight fever for a further week. The provisional diagnosis was infective hepatitis.

An X-ray examination of the chest showed it to be normal. Microscopic examination of specimens of the urine showed 10 red blood cells per high-power field. The urinary urobilinogen level was markedly increased, and Fouchet's test for bile gave positive results. The white blood cell count was 9500 per cubic millimetre, with a normal differential count. Urine was injected into guinea-pigs, but no leptospirosis was produced.

On July 3, liver function tests yielded the following results: icteric index, 35 units; serum alkaline phosphatase content, 28 King-Armstrong units; zinc sulphate turbidity, 2·5 units; thymol turbidity, two units; thymol flocculation test, negative result. By July 9, liver function tests yielded the following results: icteric index, 15 units; serum alkaline phosphatase content, 30 King-Armstrong units; zinc sulphate turbidity, six units; thymol turbidity, 12 units; thymol flocculation test, positive result. On August 3, results were as follows: serum protein content, 6·2 grammes *per centum*; icteric index, two units; serum alkaline phosphatase content, six King-Armstrong units; zinc sulphate turbidity, 6·5 units; thymol turbidity, 7·5 units; thymol flocculation test, positive result. On July 6, the complement fixation reaction for *Coxiella burneti* gave a titre of 1/512. On August 9, the reaction was anti-complementary. Agglutination tests for leptospirosis, typhus and typhoid gave negative results.

At no time were there any respiratory symptoms or signs. He was discharged from hospital fully recovered on August 3. No specific treatment was given.

CASE II.—The patient was a male, aged 50 years. He had worked for 35 years at the Ross River meatworks, where, at the time of the onset of his illness, he was employed wiping down cattle on the killing floor. He was admitted to the Townsville General Hospital on July 21, 1956, with a history of nausea and malaise for the preceding 11 days, associated with pain in the right hypochondrium. He was noticed to be jaundiced, and the liver was enlarged and tender. Fever was present from August 1 to 5, and a transient scanty maculo-papular rash appeared on the abdomen. The provisional diagnosis was infective hepatitis.

An X-ray examination of the chest gave normal findings. The white cell count was 11,600 per cubic millimetre, the differential count showing a slight polymorphonuclear leucocytosis. On July 25, liver function tests yielded the following results: serum bilirubin content, five milligrammes per 100 millilitres; serum alkaline phosphatase content, 32 King-Armstrong units; zinc sulphate turbidity, six units; thymol turbidity, 12 units; thymol flocculation test, positive result. By August 31, liver function tests showed: icteric index, three units; serum alkaline phosphatase content, 10 King-Armstrong units; zinc sulphate turbidity, six units; thymol turbidity, seven units; thymol flocculation test, positive result. On August 15, the Paul-Bunnell test gave a negative result. The complement fixation test for *C. burneti* showed the following titres; first specimen (August 7), 1/128; second specimen (September 3), 1/128; third specimen (November 9), 1/256; fourth specimen (March 27, 1957), 1/64. Agglutination tests for leptospirosis, typhus and typhoid all gave negative results.

He was discharged from hospital perfectly well on August 8, 1956. No specific treatment was given.

CASE III.—The patient was a male, aged 50 years. He was working as a railway porter at Ingham, but also had a part-time job milking cows for three months before the onset of his illness. He was admitted to the Ingham Hospital on May 17, 1957, complaining of anorexia, and it was noticed that he was febrile and jaundiced. His jaundice cleared in two weeks, but a low-grade pyrexia with a temperature of 100° F. to 101° F. persisted, and he was transferred to the Townsville General Hospital on June 27 for further investigation. An X-ray examination of his chest at Ingham showed it to be normal, and a film taken on his arrival in Townsville was also normal. His temperature remained at 99° F. until July 6, when his fever disappeared. Examination of his urine showed no albumin or sugar, and microscopic examination showed it to be normal. Physical examination revealed no abnormality. He was not jaundiced and his liver was not palpable.

On July 8, liver function tests yielded the following results: serum bilirubin content, 0·2 milligramme per 100 millimetres; alkaline phosphatase content, eight King-Armstrong units; thymol turbidity, six units; thymol flocculation test, positive result; zinc sulphate turbidity, seven units; blood urea content, 18 milligrammes per 100 millilitres; hæmoglobin value, 13·9 grammes *per centum*; white cell count, 6000 with a normal differential count; erythrocyte sedimentation rate, 40 millimetres in one hour (Westergren). On June 25, the complement fixation test for *C. burneti* gave a titre of 1/512. When this test was repeated on July 17, the titre was 1/256. Leptospiral, typhus and typhoid agglutination tests gave negative results.

At no time did this patient have any respiratory symptoms. He received no specific treatment.

CASE IV.—The patient was a male, aged 31 years. He was employed as a meatworker near Townsville. He was admitted to the Townsville General Hospital on October 3, 1957, with a history of illness for two weeks, his main symptom being severe headache. For one week he had had anorexia and a slight cough. Physical examination of the patient showed no abnormality apart from some tenderness and rigidity in the epigastrium. By October 10, the liver was palpable and tender three fingers' breadth below the right costal margin. His temperature was 103° F. on his admission to hospital, and he had rigors. The temperature ran a remitting course between fever peaks of 102° F. and 101° F. until October 16, when its subsidence coincided with the commencement of therapy with oxytetracycline, 250 milligrammes every six hours. He had earlier been treated with penicillin with no therapeutic effect, as the physician who admitted him to hospital made a provisional diagnosis of diaphragmatic pleurisy. With the subsidence of his fever all his symptoms vanished, and he was well when discharged from hospital on October 23. At no time was he jaundiced.

On October 3, an X-ray examination of his chest showed it to be normal. On October 9, the white cell count was 9000, neutrophils being 77%, lymphocytes, 20%, and monocytes 3%. On October 11, liver function tests yielded the following results: serum bilirubin content, 0·3 milligramme per 100 millilitres; alkaline phosphatase content, 16 King-Armstrong units; thymol turbidity, 10 units; thymol flocculation test, positive result; zinc sulphate turbidity, 11 units. On October 9, a complement fixation test for *C. burneti* showed a titre of 1/64. By October 22, the titre had risen to 1/512, and on November 25 it was 1/512.

Discussion.

It will be noted that the provisional diagnosis in the first three cases was infective hepatitis. In Case I, although there was no clinical reason to doubt a clinical diagnosis of infective hepatitis if the patient was viewed as an individual, it was known that no patients with infective hepatitis had been admitted to the Townsville General Hospital for over a year, and the possibility of some other cause for his hepatitis was considered. Further doubt was also cast on the diagnosis when in the battery of liver function tests it was noticed that on July 3, 1956, the serum flocculation tests gave normal results, but when the results became abnormal on July 9, the zinc sulphate turbidity test gave normal results in the presence of abnormal thymol turbidity and an abnormal result to the thymol flocculation test. In infective hepatitis, Sherlock (1955) states that "the Zinc Sulphate test is positive early in the disease and the Thymol Turbidity later". While this is probably by no means a hard and fast rule, it is a good guide, and in any undoubted epidemics of infective hepatitis seen in this area, the zinc sulphate test usually gives positive results early. At that time it was not realized that "Q" fever could simulate infective hepatitis, and serological tests were performed to exclude possible leptospirosis. It was fortunate that, as a routine, the Laboratory of Microbiology and Pathology in Brisbane performs a battery of serological tests, including the complement fixation reaction for *C. burneti*, on all sera submitted to it for examination in cases of fever of unknown origin. Because of this test the high titre to *C. burneti* was discovered. When the patients in Cases II and III presented in a similar way to the patient in Case I, with similar serum-flocculation findings, it was realized that they could also have "Q" fever, and the high titres confirmed this possibility. In these three cases there was no evidence of involvement of any organ other than the liver, and the clinical presentation was exactly as in infective hepatitis. The three patients all presented jaundiced, which is apparently unusual, as in most accounts of the disease the jaundice develops late. In

Case IV there was no confusion with infective hepatitis on the patient's admission to hospital, but the combination of headache, anorexia, fever and a tender liver could easily suggest the pre-icteric stage of infective hepatitis. In this case it is interesting that the zinc sulphate turbidity test gave weakly positive results, eight units being allowed as the upper limit of normal for this test.

There can be little doubt that all four cases were "Q" fever. The titres found in the complement fixation test are such that Derrick (1957), who discussed them with Tonge (1957), is of the opinion that such high titres are not seen except in "Q" fever. The possibility of coincidental infective hepatitis is also very unlikely. Records of the Townsville General Hospital show that no patients with infective hepatitis were admitted in the year 1956, and only one patient was admitted to December, 1957; that case did not occur locally, the patient being a child transferred from Ayr, 60 miles away. The last epidemic of infective hepatitis occurred in the Townsville area in 1953-1954; it mostly affected people in the suburb of Garbutt and also nurses in the Townsville General Hospital. Three patients were admitted in 1952, 19 patients in 1953, 12 patients in 1954 and three patients in 1955. The occupation of the patients in Cases II, III and IV was such that "Q" fever constituted a well recognized occupational hazard. It is interesting to note that there was no epidemic of "Q" fever in the area at the time. No other patients were admitted to the Townsville General Hospital in 1955, 1956 or 1957, and the disease does not appear to be common in the area. If it occurred it would certainly be detected, as the complement fixation test for *C. burneti* is always performed as a routine in all cases of fever of unknown origin.

Conclusion.

The inference is that "Q" fever hepatitis may be much more common than is usually recognized, and if all cases of hepatitis were investigated with this possibility in mind, many cases would be discovered masquerading as infective hepatitis. Wespi (1957) was able to find reports of only 17 jaundiced patients in the literature, his own patient making the eighteenth. The finding of three jaundiced patients with "Q" fever within 15 months in a general hospital with approximately 350 beds may give some idea of the true incidence of the condition. From the histories of the foregoing cases it is easy to see how confusion with infective hepatitis could arise, and this possibility is also mentioned by Clark *et alii* (1951), by Ludwig (1956) and by Wespi (1957). In these three cases associated with jaundice, the symptomatology and clinical course of the disease were such as might be expected in infective hepatitis. From these cases it is apparent that an occupational history giving contact with cattle is important; even the patient in Case I, as he is a railway fettler, could have contact with livestock being railed to meatworks. Again, if in a suspected case of infective hepatitis the zinc sulphate turbidity test gives negative or only slightly positive results, possible "Q" fever hepatitis should be considered. Because of the widespread epidemics of infective hepatitis which occurred during and since the war of 1939 to 1945, it is possible that, owing to familiarity with this condition, cases of "Q" fever hepatitis may easily be confused with it.

If "Q" fever hepatitis is established, then there are therapeutic possibilities in the use of the tetracyclines, though naturally there is as yet no statistical evidence that the hepatic form of the disease responds as well to antibiotic treatment as the other forms of the disease.

Summary.

Four cases of "Q" fever hepatitis are described, in three of which jaundice occurred. These cases were sporadic and did not occur in the course of a human epidemic of the disease. In the three cases associated with jaundice, the mode of presentation made clinical differentiation from infective hepatitis impossible. There was no epidemic of infective hepatitis in the area at the time. Possible differentiating points between the two conditions are discussed.

Acknowledgements.

I should like to thank Dr. E. H. Derrick, Deputy Director of the Queensland Institute of Medical Research, for his generous advice and help with references. The serological tests were performed at the Queensland Laboratory of Microbiology and Pathology, Brisbane, under the direction of Dr. J. H. Tonge. The liver function tests were performed at the Commonwealth Health Laboratory, Townsville, initially under the direction of Dr. A. Ivanov and latterly under that of Dr. R. A. Rimington.

References.

CECIL, R. L., LOEB, R. F., Editors (1955), "A Textbook of Medicine", 9th Edition, Saunders, Philadelphia. 102.

CLARK, W. H., LENNETTE, E. H., RAILSBACK, O. C., and ROMER, M. S. (1951), "Q Fever in California: VII. Clinical Features in One Hundred and Eighty Cases", *Arch. Int. Med.*, 88: 155.

DERRICK, E. H. (1937), "'Q' Fever, a New Fever Entity: Clinical Features, Diagnosis and Laboratory Investigation", M. J. AUSTRALIA, 2: 281.

DERRICK, E. H. (1957), personal communication.

HORDER, LORD, Editor (1952), "The British Encyclopædia of Medical Practice", 2nd Edition, Butterworth, London, 12: 390.

HUNTER, D., Editor (1956), "Price's Textbook of Medicine", 9th Edition, Oxford University Press, London: 302.

LUDWIG, H. (1956), *"Q-Fieber-Hepatitis", Schweiz. med. Wchnschr.*, 86, Suppl.: 1052.

MACGRAITH, B. (1956), "Q Fever", in Hunter, D., *loco citato.*

MEGAW, J. W. D. (1952), "Typhus Fevers and other Rickettsial Fevers", in Horder, Lord, *loco citato.*

SHERLOCK, S. (1955), "Diseases of the Liver and Biliary System", Blackwell, Oxford: 258.

SNYDER, J. C. (1955), "Q Fever", in Cecil, R. L., and Loeb, R. F., *loco citato.*

TONGE, J. I. (1957), personal communication to E. H. Derrick.

WESPI, H. (1957), *"Ein Fall von Q-Fieber-Hepatitis", Schweiz. med. Wchnschr.*, 87: 90.

HERPANGINA: ULCERATIVE PHARYNGITIS WITH THE ISOLATION OF COXSACKIE VIRUS.

By IAN JACK AND R. A. CHENOWETH,

From the Department of Pathology, Sporting Globe-3DB Research Unit, Royal Children's Hospital, Melbourne.

HERPANGINA was described by Zahorsky in 1924. The disease is characterized by a sudden onset, with fever, a sore throat and difficulty in swallowing. Papules or vesicles are found on the walls of the pharynx, the pharyngeal pillars, tonsils, uvula or soft palate. They are grey or white in colour and one to two millimetres in diameter. The disease is infectious. Recovery from systemic symptoms usually occurs in three days with disappearance of the local lesions in six days. The disease usually runs a benign course and complications are rare.

Ulcerative gingivo-stomatitis, due to the *herpes simplex* virus, follows a similar course, but gingival lesions and involvement of the cervical lymph nodes are more common in this disease.

Twenty-seven years after Zahorsky's clinical description of the disease, Huebner *et alii* (1951) incriminated group A Coxsackie virus as the causal agent. Since then this finding has been confirmed in many parts of the world. This communication records three cases occurring in Melbourne from which Coxsackie virus has been isolated.

Reports of Cases.

CASE I.—The patient, a female aged 16 months, was brought to the Royal Children's Hospital, Melbourne, on November 4, 1954. For two or three days before she had had a running nose, and on the day before she had had five bowel actions. She had been well prior to this.

Examination revealed a normal temperature and pulse rate. The only positive finding was the presence of vesicles on the anterior faucial pillars on both sides, about eight in number and one to three millimetres in diameter, with an erythematous base. A diagnosis of herpangina was made, and a blood sample, throat swab and specimen of fæces were sent for virus examination. A virus identified as Coxsackie A was isolated from the throat swab.

Seven days later the child had recovered completely. No sign of the local lesions remained. It was discovered on the second visit that at the migrant camp, where this family lived, there had been many sore throats in the previous week.

CASE II.—The patient, a female of Italian birth, aged two years, was first seen on November 14, 1954. For the preceding 24 hours she had been feverish and one hour before her admission to hospital had had a convulsion.

Examination revealed a temperature of 104·1° F. and the presence of vesicles on the anterior faucial pillars and tonsils. No other abnormality was present. A blood sample, throat swab and specimen of fæces were sent for virus examination, and from the fæces a Coxsackie A virus was isolated.

Three days later the vesicles were partially healed but she was still flushed and irritable. Her next appointment was not kept.

CASE III.—The patient, a male aged 18 months, was seen on December 3, 1954. For 12 hours he had been feverish, with a slight cough and loss of appetite. He had vomited once.

Examination revealed a temperature of 99·2° F. and vesicles on the fauces. No other abnormality was present. A blood sample, throat swab and specimen of fæces were sent for virus examination, and from the throat swab a Coxsackie A virus was isolated. Examination three days later showed improvement in general condition and disappearance of the vesicles.

Isolation of the Virus.

For preference, virus isolation was attempted from the site of infection rather than from the fæces. Only two of the three throat swabs yielded a virus; virus was isolated from the fæces of the third patient.

Specimens were inoculated into one-day-old suckling mice by the intraperitoneal route (with the addition of 500 units of penicillin and 50 micrograms of streptomycin per dose). After 48 hours some of the mice were dead and the survivors were moribund. The survivors were killed, skinned, eviscerated, ground with sterile alundum and emulsified to produce a 20% suspension in 0·5% gelatin-saline.

This first passage material was inoculated into a further litter of suckling mice, and onto the chorio-allantoic membranes of 12-day-old embryonated hen's eggs. As no pocks were produced on the membranes in two to three days, although the second group of mice were already showing signs of infection, it was concluded that the agent was not *herpes simplex* virus.

In the case of the virus isolated from the throat of the patient in Case I, one mouse, which had survived until the fifth day but which showed a marked hind leg paresis, was killed and sections were prepared from leg muscles, brain, pancreas and fat pads. On histological examination, the skeletal muscles were found to be abnormal, showing a generalized myositis in which hyaline swelling and loss of striations were present. This suggested that the agent was a Coxsackie virus of the A group, since there was no involvement of the central nervous system and the myositis was not focal. No attempt was made to passage the virus intracerebrally before the agent was submitted to Mr. D. C. Dorman at Prince Henry Hospital, Sydney, for typing. He confirmed it as a group A Coxsackie virus, and identified it as A4 (Dalldorf).

In the meantime two further isolations had been made, from the throat swab of the patient in Case III, and from the fæces of the patient in Case II. These were found to be neutralized by mouse antiserum prepared against the Case I virus, and were accordingly classified as A4 also.

Examination of Serum.

Paired sera were obtained only from the first patient. Table I shows the neutralizing antibody titres thus obtained by the method adopted by Stanley (1951) in which a dose of 100 LD_{50} of virus is incubated with an equal volume of serum dilutions at room temperature for 60 minutes prior to inoculation to day-old suckling mice.

The patient in Case I, from whom the virus was isolated from the site of infection, and in whom a significant rise in titre of specific neutralizing antibody occurred during convalescence, is convincing evidence of the relationship of the virus isolated to the disease.

The failure to isolate virus from the throat swab of the patient in Case II, although virus was isolated from the fæces, meant that final diagnosis was dependent on detection of an antibody rise during convalescence. Unfortunately, a convalescent serum sample was not received. However, the similarity of the clinical picture with that of Case I and the identity of the virus suggest that it was related to the disease. The patient in Case III, from whom the virus was recovered in the throat swab, also lacked a second serum sample, but this is partly compensated by isolation of the virus from the throat.

TABLE I.

Titres of Neutralizing Antibody Against Coxsackie A4 Virus in Paired Sera (Case I).

Serum Taken.	Number of Mice Dead by Twelfth Day.			PD_{50}.
	Serum Dilutions.		Controls (50 LD_{50}).	
	1 : 80.	1 : 320.		
First day ..	6/6	7/7	7/7	80
Seventh day ..	0/7	4/6		<240

Discussion.

Overseas reports since 1951 have implicated group A Coxsackie viruses as the causative agents of herpangina. The disease also occurs in Australia. That the disease is not new is seen from two viewpoints. Clinically, McLorinan has known the condition for some years in Melbourne by the name used by the older nose and throat textbooks, "*herpes pharyngis*". On the laboratory side, Stanley *et alii* (1953) have reported the presence of group A antibodies in serum samples obtained from all States. Further, types A6 and A8 have been isolated in our laboratory from cases of meningo-encephalitis.

The occurrence of only a limited number of cases does not accord with the epidemic nature of herpangina. However, one of the cases came from a migrant hostel where "sore throats" had been epidemic. The isolation of the same virus type from three different areas suggests that it may have been widespread at the time.

Despite the name herpangina the disease is unrelated to the gingivo-stomatitis of *herpes simplex*. The clinical and ætiological differentiation of these two conditions is described in the well-illustrated article of Parrott *et alii* (1954).

Summary.

Three cases of clinical herpangina are described. The isolation of type A4 Coxsackie viruses is reported.

Acknowledgements.

We thank Mr. D. C. Dorman, of Prince Henry Hospital, Sydney, and Professor N. F. Stanley, formerly of Prince Henry Hospital, Sydney, for typing the virus. Dr. A. L. Williams of the Royal Children's Hospital, Melbourne, examined the mouse tissues.

References.

HUEBNER, R. J., COLE, R. M., BEEMAN, E. A., BELL, J. A., and PEERS, J. H. (1951), "Herpangina: Etiological Studies of a Specific Infectious Disease", *J.A.M.A.*, 145 : 628.

McLORINAN, H., personal communication.

PARROTT, R. H., WOLF, S. I., NUDELMAN, J., NAIDEN, E., HUEBNER, R. J., RICE, E. C., and McCULLOGH, N. B. (1954), "Clinical and Laboratory Differentiation Between Herpangina and Infectious (Herpetic) Gingivostomatitis", *Pediatrics*, 14 : 122.

STANLEY, N. F., DORMAN, D. C., and PONSFORD, J. (1953), "Studies on Australian Strains of Coxsackie Virus (Group A and B)", *Australian J. Exper. Biol. & M. Sc.*, 31 : 21.

STANLEY, N. F., HATES, L., and DORMAN, D. C. (1951), "Suckling Mouse Viruses in New South Wales: Their Relationship to the Coxsackie Group of Viruses", *Australian J. Exper. Biol. & M. Sc.*, 29 : 367.

ZAHORSKY, J. (1924), "Herpangina (A Specific Infectious Disease)", *Arch. Pediat.*, 41 : 181.

A DELAYED DIAGNOSIS OF BUNG EYE.

By DAVID J. LEE,
Entomologist, School of Public Health and Tropical Medicine, University of Sydney.

IN a paper presented to the Royal Society of New South Wales, Dr. Eustace W. Ferguson (1915) discussed the occurrence of bung eye in the Sydney area. He stated:

> I have reason to think, however, that Bung Eye is probably more prevalent in the neighbourhood of Sydney than is generally thought. Over sixty cases occurred among the patients at Rydalmere Hospital for the Insane during three months in the beginning of the summer of 1912-1913. I have also been informed that Bung Eye was exceedingly common among the men engaged in building the dam on the Hawkesbury River, on the mainland opposite Milson Island, in a locality where these flies have been taken on several occasions. Bung Eye also occurs at Mount Irvine in the Mount Wilson district, a locality whence these flies have been obtained.

The flies referred to by Ferguson were species of *Spaniopsis*, a blood sucking genus of the family Leptidæ, allied to the March flies (Tabanidæ). Ferguson also stated, in answer to the suggestion that these flies might have a causal connexion with bung eye, that he did not think that this was likely. In this Ferguson was correct, and there is no suspicion that *Spaniopsis* causes swelling of the eyelids.

However, Ferguson's citation of cases of bung eye in the Sydney area in the early summer does link up with recent observations on another biting fly, namely *Leptoconops stygius* Skuse, a sandfly belonging to the family Ceratopogonidæ. *L. stygius* was first described in 1889 from specimens taken at Woronora. Up to 1947 (Lee, 1948) the original record, together with additional specimens from Fitzroy Falls, comprised all that was known of this species. In the last 10 years many thousands of these flies have been captured by the writer and his colleagues, and much more is known of its distribution and seasonal occurrence.

L. stygius occurs characteristically during the first fortnight of November throughout many of the sandstone gulleys of the Hawkesbury River system. The earliest records for the season occur at the beginning of October and the latest in January. However, in most seasons high population levels occur only in early November. In bushland areas these flies can be noticed swarming around the head, and on hot, humid days they will attack and bite, especially along the hair line or on the scalp. The attack is usually experienced in gullies, but it does, on occasion, also take place on ridges.

No effect other than transient irritation seems to follow bites on the scalp, but if the bite is on the face the area surrounding the eye is usually attacked and the sequelæ may take the alarming form of bung eye. Both the upper and lower eyelids become œdematous, and the swelling completely closes the eye. No pain is experienced and the actual site of the bite may or may not be obvious.

One subject, an adult female, has been affected in this way on six occasions in five years during the first half of November. On one occasion *L. stygius* was taken from the eyelid while biting, and the swelling commenced within the next two hours and increased until the eye area was the size of half a golf ball and the eye completely closed. This took about 12 hours, and then the swelling gradually subsided over the next three to four days. For this season the earliest case was recorded in a young girl on October 17, 1957. This followed early sightings of *L. stygius* a week previously.

Ferguson's (1915) records, stressing early summer and areas within the Hawkesbury River system, are highly suggestive, and it seems more than likely that *L. stygius* was involved in these early cases. The Rydalmere cases remain doubtful, as the environment is not typical of the habitats exploited by *L. stygius*. Nevertheless, complaints of sandfly activity have been received from nearby Dundas at times when *L. stygius* was known to be at its peak, but no specimens have been examined.

L. stygius is also known to attack poultry during the daytime; in this case the comb and wattles are attacked. It also attacks horses; on these animals it bites within the ear and produces a hard thickening of the ear itself.

Summary.

L. stygius is a diurnally active blood-sucking sandfly, occurring in the early summer with a peak abundance in the beginning of November, found over a wide area of the Hawkesbury River system from the coastal inlets to the Blue Mountains. It attacks man, other animals and poultry. In man, if the site of the bite is in the vicinity of the eye, it is likely to cause an acute, painless œdema of the eyelids. Ferguson's early records of bung eye in the Sydney area are now considered to have been caused by *L. stygius*. This local condition has no known relation to bung eye occurring in inland Australia.

References.

FERGUSON, E. W. (1915), "Description of New Australian Blood-Sucking flies Belonging to the Family Leptidæ", *J. Roy. Soc. N.S.W.*, 49: 233.

LEE, D. J. (1948), "Australian Ceratopogonidæ (Diptera, Nematocera): Part II. The Leptoconops Group of Genera", *Proc. Linn. Soc. N.S.W.*, 72: 332.

Reviews.

The Peter T. Bohan Memorial Lectures on Medicine. First Series; 1957. Lawrence: University of Kansas Press. 9¼" × 6", pp. 152, with four illustrations. Price: $3.00.

ONE cannot order a consistently excellent series of memorial lectures by simply providing funds and an auditorium. Even the annual Harveian, Lumleian and similar lectureships provided by the Royal College of Physicians of London are not uniformly of the highest quality. The same result is manifest in a recent collection of the Peter T. Bohan Lectures delivered as an annual memorial at the University of Kansas. Seven lectures by prominent American physicians and surgeons have been published in an attractive volume in memory of a former popular and gifted teacher of this campus. The first is entitled "Pernicious Anæmia from Addison to Folic Acid" by Dr. Russell Haden. This is a simple and interesting account of the historical contributions by many great men during the past one hundred years. It is not a profound or highly technical description, but is distinctly interesting, and includes a full bibliography.

Walter Alvarez, sometime gastro-enterologist at the Mayo Clinic, gave the next published lecture in 1948, and called it "Some Functional Distress in the Abdomen". He is a good and experienced writer, but on this occasion produced a rambling, superficial and rather distressing description of minor abdominal complaints for which he offers explanations often naïve and original. Dr. John King lectured in the following year, choosing to discuss the role of minor focal infection in the genesis of coronary disease and hypertension, a theory with few supporters and difficult to substantiate.

"Urology and Internal Medicine" is a lecture by Dr. Herman Kretschmer, in which he alerts the urologist to findings in his field which could be indicators of systemic disease. His is a useful contribution to the borderland area between urology and internal medicine. The lecturer began his career in general practice, and this is reflected in his approach to the subject; but he rather stretches out to include as many medical diseases as possible—as, for instance, when he states that the leukæmic patient frequently consults the urologist first because of hæmaturia and priapism. Dr. Claude Dixon reviewed "Surgery, General and Otherwise" in 1952. This proves to be the best offering of all, an amusing and wise dissertation on the ethics and ideals of the surgeon, which makes enjoyable reading. Dr. Paul D. White draws from "A Quarter Century of Medical Experience" for the 1953 lecture, and although half his time was passed in quotations, he reveals his good heart, his love of hard work, and his enjoyment of his personal relationships with his patients and of his association with students

and young graduates. He advocates the part-time teacher and the use of private patients for teaching, and the merit of working as one of a research team rather than as a lone investigator. The final lecture in the volume concerns "Prognosis in Coronary Artery Disease", by Dr. R. W. Scott, a thoughtful contribution on the varying arterial patterns of coronary circulation, as revealed by autopsy X-ray studies of the heart and their unpredictable contribution to the outcome of a coronary accident.

This volume of lectures is beautifully printed and easy to read. Its contents are of variable scientific and literary value, and in some cases, outmoded and questionable.

The Century of the Surgeon. By Jürgen Thorwald; 1957. London: Thames and Hudson. Sydney: Walter Standish and Sons. 8½" × 5½", pp. 428, with 66 illustrations. Price: 25s. (English).

THE nineteenth century was remarkable for the progressive acquisition, and then the practical application, of a spate of important discoveries leading to new concepts of disease and improved techniques in every department of medical science. The relief of pain, the proven safety of surgical intervention with the widening extension of its domains, a closer alliance of medicine with general science, and the promotion of positive health on a national basis, all helped to relegate professional services from the privilege of a private concern to that of a public utility, for which governments had to accept an increasing burden of responsibility. Several aspects of this sensational development are dramatically presented for popular consumption by Mr. Jürgen Thorwald in an exciting book with the attractive title "The Century of the Surgeon".

There is plenty of legitimate fiction woven into the tale, and before the glad tidings of success are finally announced the reader will need to brace himself for a few gruesome and harrowing scenes of human suffering and neglect from records of those early decades of the last century before the triumphs of anæsthesia, trained nursing and the final acceptance of Lister's antiseptic principle began to change the whole picture. Nevertheless, it must be conceded that the situations so theatrically portrayed are based on reliable evidence and even painful actuality. In fact, the material collected for each chapter must have been obtained at the cost of much time and infinite labour in careful research, as the author has succeeded in bringing to light a surprising number of unfamiliar facts relating to the earliest origins of surgical knowledge and practical procedures connected with such varied topics as renal and vesical calculi, nephrectomy, gastrectomy, appendicectomy, plastic surgery, the germ theory of disease, Cæsarean birth and puerperal sepsis.

No doubt many practitioners of medicine share with the reading public a liking for a really good story, and this book may be recommended to them with confidence as a thrilling and instructive companion for their leisure hours. The serious nature of the main theme is evidenced by the long list of sources of information quoted as having been consulted for the writing of each chapter, and by the inclusion of a bibliography of some 500 items listed at the end of the book. Also, there are many interesting plates and woodcuts interspersed throughout the text, although a few of the legends accompanying them are not strictly accurate.

Studies on the Exo-Erythrocytic Cycle in the Genus Plasmodium. London School of Hygiene and Tropical Medicine, Memoir 12, by R. S. Bray, B.Sc., Ph.D.; 1957. London: H. K. Lewis and Co., Ltd. 9½" × 7", pp. 300, with 24 illustrations. Price: £2 2s. (English).

THE microscopic demonstration of the exo-erythrocytic cycle in human malaria is one of the notable advances made in medical parasitology since the second World War, which was itself a period of considerable advancement in the knowledge of malaria. Stages similar to these were already known in avian infections and had been forecast by several workers, notably Fairley and his colleagues, for human infections. This new knowledge led to a much fuller understanding of the natural history of malaria infection and gave a rational basis for chemotherapy—especially that directed towards the eradication of relapsing vivax infections.

Three cycles of development are now established in the life history of malaria parasites: exo-erythrocytic, erythrocytic (asexual) and sexual. In this monograph the author has made a valuable contribution to parasitology, in the form of an extensive critical review of the literature on the exo-erythrocytic stage of development of mammalian, avian and saurian plasmodia. The discussion ranges over morphology, biochemistry, response to chemotherapy, and immunity, indicating the broadened scope of parasitology in recent years. He also presents results of his own work.

The careful historical survey of the subject apportions credit to those responsible for the accumulation of this knowledge; there was a controversy ten years ago concerning priority of discovery, which threatened to be a repetition of the Ross-Italian dispute. Much of this work on mammalian parasites has been done at the London School of Hygiene and Tropical Medicine. It is fitting that this report should be published in the monograph series of that school; but it is a pity that some of the coloured plates from the original papers have not been included.

In America, the parenchymal cells of the liver have not been universally accepted as the site of development of the exo-erythrocytic stage of mammalian malaria. One of the conditions of acceptance laid down by Huff elicits an impatient response on the part of the author.

It has been known for a number of years that the family Plasmodiidæ was due for revision. The author does not fully agree with the classification suggested by Garnham and presents a subdivition of the family into two genera: *Plasmodium* and *Hæmamœba*. This is probably not the last word on this subject, but at least it does not disturb the nomenclature of the medical parasitologist.

Schaudinn's observation of a sporozoite of *P. vivax* entering a red blood cell, reported in 1903, concealed the exo-erythrocytic stage of development for 30 years, so great was his reputation as a parasitologist. How surprised he would be to know of the intricate development of that sporozoite before the red cell is actually invaded!

A Manual of Human Anatomy. Volumes I to IV, by J. T. Aitken, M.D., G. Causey, M.B., F.R.C.S., J. Joseph, M.D., M.R.C.O.G., and J. Z. Young, M.A., F.R.S.; 1956. Edinburgh and London: E. and S. Livingstone, Limited. Volume I: "Thorax and Upper Limb", 8½" × 5½", pp. 180, with illustrations. Price: 14s. Volume II: "Head and Neck", 8½" × 5½", pp. 188, with illustrations. Price: 16s. Volume III: "Abdomen", 8½" × 5½", pp. 144, with 29 illustrations. Price: 12s. 6d. Volume IV: "Lower Limb", 8½" × 5½", pp. 125, with illustrations. Price: 12s. 6d. Volume V: "Central Nervous System", by J. T. Aitken, M.D., D. A. Shell, B.Sc., and J. Z. Young, M.A., F.R.S., 8½" × 5½", pp. 148, with 40 illustrations. Price: 12s. 6d.

THIS set of volumes is a new attempt, on the part of five British authors, to deal with the problem of the heavy load involved in the traditional requirements for the final examination in anatomy for medical undergraduates. The authors seek to deal with the problem along the traditional lines of dissection, but with a great deal of detail omitted. They have attempted to produce what is both a manual of dissection and a manual of anatomy intended to guide the student "as to the extent of knowledge expected of him in the second medical examination". Further, an attempt is made to guide the student systematically into the profitable fields of practical and functional anatomy.

In assessing the value of a dissecting manual, one is handicapped by not knowing what, if any, text-book is prescribed for reference, and to what extent used; whether the student is to dissect the body more than once; whether he has had the opportunity of preliminary study of dissected parts; and so on. However, on the assumption that Volumes I to IV of this manual are to be used by the inexperienced dissector, it is doubted whether the instructions are detailed enough to provide an adequate guide for the average student, and this doubt is strengthened by the relative paucity of clear and accurate drawings to supplement the text and illustrate the instructions for dissection. Again, in several instances, one fears that the "bold stroke" method of dissection, while providing a challenge to the more enterprising dissector, may more often mean the loss of easily acquired and important knowledge—as, for example, in removing the sterno-mastoid muscle, in dissecting the thenar region, and in clearing the anterior aspect of the thigh down to the deep fascia. However, in the selection of the "important" and the elimination of "needless detail", opinions must necessarily vary considerably.

The sections on practical work in osteology, surface anatomy and functional anatomy may be a useful guide for students in schools where such work is not already planned as part of their course; and the separate treatment of the lymphatic drainage of each region, though necessarily brief, will emphasize to the student the importance of this aspect of the subject.

It is when we come to the qualities of Volumes I to IV as a manual of anatomy and as a basis for future clinical work that we unfortunately find a good deal to criticize. In the first place, the "pruning" appears to be excessive and

at times uneven. The treatment of several regions and organs is far too meagre. Further, there appear to be many errors both in the text and in the figures.

The meagre treatment of important topics is well illustrated in the brief and sketchy section on the venous drainage of the leg (Volume IV, pages 95-97), to which Figure 36 lends little support; varicose veins and their complications contribute a large percentage of patients attending out-patient departments. A similar sketchiness is found in other important matters—for example, in the relations of the œsophagus, in the blood supply of the alimentary tract. Here again, of course, there is room for individual judgement.

Some of the several features in the text to which exception can be taken are: the complete omission of any reference to the frequently anomalous innervation of the thenar muscles (Volume I, pages 135-136); the demonstrably erroneous statement that gleno-humeral abduction and lateral rotation of the scapula each contribute about 90° of the total range of abduction (Volume I, page 101); the traditional but erroneous account of the anatomy of the inguinal region (Volume III, pages 7-13).

The figures are clearly drawn, but regrettably they contain many errors. Of these, some of the more serious are: Volume I, Figure 8—the phrenic and vagus nerves are incorrectly related; Volume I, Figure 17 (also Volume II, Figure 26)—this is misleading in the relation of the axillary artery to the large nerves; Volume II, Figure 8—there is inaccuracy in the attachment of the temporalis muscle; Volume IV, Figure 21—there is a misleading representation of the lateral aspect of the ankle joint region; Volume IV, Figure 25—the division of the posterior tibial nerve is far below the usual level.

To sum up, it is our opinion that in their present form Volumes I to IV are not sufficiently detailed and adequately illustrated to act as a guide to the dissector; and that considerable correction and expansion are needed before they could be regarded as a satisfactory manual of anatomy.

Volume V, on the central nervous system, deals with somewhat different problems of dissection and presentation. Here the result is more successful. There are several good features, including the clearly drawn and simplified cross-sections of the brain stem. It is suggested that this volume would be improved by the inclusion of clearly drawn diagrams of the sensory, auditory and visual pathways, by the more precise definition of such cortical areas as the motor cortex, the visual area and the like, and by improvements in some of the figures—for example, Figures 34 and 38. Again it is doubted whether the contents of this volume are really adequate in scope and detail as a preparation for future clinical work.

The volumes are well printed, light and easy to handle.

Advances in Tuberculosis Research. Volume 8: BCG—A Discussion of Its Use and Application. Edited by Dr. Hans Birkhäuser and Dr. Hubert Bloch; 1957. Oxford: Blackwell Scientific Publications. 9¼" x 6¾", pp. 328, with five illustrations. Price: 60s. (English).

THE eighth volume in the series "Advances in Tuberculosis Research" is devoted entirely to a discussion of B.C.G., its use and application. The editors point out that they have "attempted to emphasise the controversial questions concerning BCG". They express happiness at "obtaining the collaboration of a number of experienced and competent authors". This is an under-statement indeed. Most of the leading workers of the last 20 years in this field, from France, Scandinavia, Britain, the United States and South America, have contributed their ideas and experiences; and B.C.G.'s stoutest opponent, J. A. Myers, of Minneapolis, presents a characteristically forthright summary of views opposing B.C.G.

Variations from the classical methods of preparing vaccines are discussed by van Deinse, of the Institut Pasteur, by Rosenthal, of Chicago, and by the late A. Q. Wells, of Oxford. Birkhaug presents a wealth of information on methods of vaccination, and weighs the advantages of the intracutaneous and percutaneous (multiple puncture) methods. De Assis describes South American experiences with "indiscriminate BCG vaccination by mouth" without preliminary tuberculin testing or X-ray examination.

The results of B.C.G. vaccination in human populations under various epidemiological conditions are described by those who have been primarily responsible for the best known trials and major applications of the vaccine in Europe, Asia and America. Aronson's account of his own and other work in the United States is impressive as a careful and reasoned assessment of a great deal of painstaking work. The Medical Research Council's controlled trial of B.C.G. and Vole bacillus vaccine, which is still going on in England, is discussed by D'Arcy Hart, Pollock and Sutherland, who are responsible for it. Both in the first progress report (1956) and in this summary, the results serve to answer the more serious objections to B.C.G., raised by Myers and others. Finally, Professor Mitchison discusses the possible benefits and dangers of prophylactic chemotherapy, either as an alternative approach to tuberculosis control or as a complement to a B.C.G. programme.

The volume does great credit to its able editors for their success in presenting almost every facet of the "growing fringe" of knowledge about specific protection against tuberculosis. Despite the diversity of disciplines and views represented, a unity has been achieved. This is not just a series of papers. It is a book about B.C.G. in the 1950's—an authoritative book, and one which should be read by every worker and teacher in the field.

Headache: Diagnosis and Treatment. By Robert Ryan, B.S., M.D., M.S., F.A.C.S.; Second Edition; 1957. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical), Limited. 8½" x 5½", pp. 424. Price: £3 14s. 3d.

THIS is certainly an excellent introduction to the subject of headache and its causes. First, there are diagrams showing the nerve pathways involved in head pain and also diagrams of the extracranial and intracranial blood vessels and the nasal sinuses. Then there are drawings illustrating the state of the nerve and the artery in the vasoconstrictive and vasodilatory phases of vascular headache. Furthermore, there is a representation of the vascular œdema phase of vascular headache. Head pain may be due to the tissues covering the cranium, to the cranial periosteum or to intracranial structures including the trigeminal glossopharyngeal and vagus nerves, the first three cervical nerves, the cerebral and dural arteries, parts of the dura at the base of the brain and the great venous sinuses and their venous tributaries from the surface of the brain. Most headaches are believed to be vascular in origin, and due to vasodilatation and the excitation of nerve endings (sensory) on or near the wall of the blood vessel. Vasoconstriction and sometimes œdema follow vasodilatation. Ryan discusses treatment of the attacks of headache, and mentions "Cafergot" and "Cafergot P.B.", the latter a suppository containing ergotamine tartrate two milligrammes, caffeine 100 milligrammes, pentobarbital sodium 60 milligrammes, and "Bellafoline" 0·25 milligramme. He claims considerable success with these preparations and with histamine given intravenously. Of tranquillizing drugs Ryan does not write very favourably, and he mentions the possible toxic effects. The differential diagnosis of the cause of headache is discussed at length. The importance of careful history-taking and examination of the patient is stressed. Many varieties of headache are discussed individually—infections, migraine, abdominal migraine, ophthalmic migraine, tension headache, psychogenic headache, headache due to ear, nose and throat and eye diseases, and many other varieties. There is a chapter on trigeminal neuralgia, in which he quotes many authors and a variety of treatments, indicating perhaps how unsatisfactory the treatment of this complaint is. There is no doubt that the types of headache described are mostly well known, though not all would accept some of the varieties, nor would all agree with the treatments advised. However, there is much of value and much food for thought in this book.

Clinical Electrocardiography: The Spatial Vector Approach. By Robert P. Grant, M.D.; 1957. New York, Toronto and London: The Blakiston Division, McGraw-Hill Book Company, Incorporated. 8½" x 5½", pp. 240, with 95 illustrations. Price: $7.50.

As the electrical activity of the heart takes place in three dimensions, and each beat produces activity of definite duration and magnitude, it is surprising that the students of electrocardiography did not from the beginning approach the subject as a problem of vectors. Although Einthoven recognized the need for the vector approach, apparently he considered it impossible to obtain the recordings in the necessary three dimensions, for his classical leads are in one plane only. The subsequent introduction of "unipolar" precordial leads and the development of "unipolar" theory further distracted attention from the vector concept. About the middle 1930's, European workers and subsequently others in the U.S.A. and Australia studied this approach to electrocardiography, chiefly with techniques using electronic equipment to record the E.C.G. as a vector pattern. However, it

is not necessary to use such equipment, as any recording made from the surface of the body is a reflection of the total cardiac electrical activity. The commonly employed twelve-lead E.C.G. can be used as effectively as the electronic display for vector purposes.

Possibly the slowness with which the vector approach has been adopted by clinical electrocardiographers is due to unfamiliarity with vector principles and unfamiliarity with display coordinates which do not include time on one of the axes.

This book presents clinical electrocardiography from a vector approach and is based on the common twelve-lead E.C.G. This treatment greatly simplifies the interpretation of changes in the E.C.G. and reduces considerably the numbers of "known" patterns which must be memorized.

The author presents his views clearly, concisely and with adequate illustrations in a pleasing production which is highly recommended to all who interpret electrocardiograms.

A Textbook of General Physiology. By Philip H. Mitchell; Fifth Edition; 1956. London, Toronto and New York: McGraw-Hill Book Company, Inc. 9″ × 6″, pp. 892, with illustrations. Price: $10.50.

THIS book was originally written to cover the syllabus of the American College Course in General Biology. It deals with a selected range of topics, including such subjects as fundamental properties of nerve and muscle, active transport across membranes, acid-base balance, blood and lymph, circulation of the blood, respiration and some aspects of intermediary metabolism and nutrition. Such a wide range of topics necessarily entails a certain superficiality of approach, and the book is thus unlikely to prove of interest either to the medical student or to the post-graduate student, since practically all the topics are more adequately dealt with in current text-books of physiology and biochemistry. However, the book may be of value to science students taking zoology or botany desiring to extend their knowledge of physiology.

Blacklock and Southwell: A Guide to Human Parasitology for Medical Practitioners. Revised by T. H. Davey, O.B.E., M.D., D.T.M.; Sixth Edition; 1958. London: H. K. Lewis and Company, Limited. 9¼″ × 5¾″, pp. 230, with three coloured plates and 119 illustrations. Price: £1 10s. (English).

IT is pleasing that the frequency of new editions of this invaluable book has apparently returned to almost pre-war regularity. Advances in parasitological research have been rapid within the last decade. Consequently, established text-books must be revised at regular intervals if they are to retain their usefulness.

In this edition the prevention of helminthic diseases has, on the whole, received more attention. While the prevention of infection with *Enterobius vermicularis* has been enlarged, it is unfortunate that the mention of the use of a piperazine derivative has been omitted, since it is so successful. The omission of the key to the intestinal species of nematodes is a loss, since many students have found this key of use in distinguishing eggs in fæces. For practical purposes this key was probably more frequently used than the key to the intestinal nematode genera, which has been retained.

The additions of *Toxoplasma* and *Isospora* to the text are worthy of note, but it is disappointing that *Pneumocystis carinii* and visceral *larva migrans* have not been included.

The new diagram of the life history of the malarial parasite is welcome, particularly since it separates so distinctly the cycles occurring in the mosquito and in man. The regrouping of the diagrams of the helminth life cycles according to the means of infection brings this section into line with an approach now becoming widely adopted. It is hoped that for a future edition the "modernizing" of these life history diagrams in the form of cycles may be considered.

The following suggested rearrangement of coloured plates I to III may add greatly to their working value. Plate I would be advantageously placed if opposite page 53, where the comparative table for intestinal protozoa is found; plate II would be better placed between chapters IX and X; plate III would be of greater value opposite page 88, where there is a relevant table, although the malarial diagrams on plate II are of greater use with this table. The success of the sixth edition of this book is assured for several reasons. It continues to be a concise work covering most of the important aspects of the subject and is still available at a relatively low cost. Once again many, including both students and teachers of medical parasitology, will be grateful to those who have laboured to produce this new edition.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Vertigo and Dizziness", by Bernard J. Alpers, M.D., Sc.D.; 1958. New York and London: Grune and Stratton. 8½″ × 5½″, pp. 128, with four illustrations. Price: $5.00.

An attempt to present the problem in a practical fashion.

"Theory and Problems of Child Development", by David P. Ausubel, M.D., Ph.D.; 1958. New York: Grune and Stratton. 9″ × 5¾″, pp. 650. Price: $12.00.

The author is from the Bureau of Educational Research, University of Illinois.

"Australia in the War of 1939-1945": Series 2 (Navy). Volume I: "Royal Australian Navy 1939-1942", by G. Hermon Gill; 1957. Canberra: Australian War Memorial. Sydney: Angus and Robertson Limited. Obtainable at all booksellers. 9¼″ × 6″, pp. 704, with many illustrations. Price: 30s.

The first of two volumes on the Royal Australian Navy.

"L'Elettroencefalogramma del Bambino Normale", by A. Fois. Pisa: Istituto di Ricerche V. Baldacci Editore. 9½″ × 6½″, pp. 140, with 101 graph readings. Price: not stated.

A discussion on the electroencephalogram of the normal infant. It is written entirely in Italian.

"Methods in Medical Research." Editor-in-Chief, James V. Warren. Volume 7; 1958. Chicago: The Year Book Publishers, Inc. 8½″ × 5″, pp. 225. Price: $7.50.

Contains articles on four subjects: chemical investigation of muscular tissues; hæmodynamic methods—heart and lungs; methods for the study of human leucocytes; methods for study of the histology and cytology of the retina.

"Ideals in Medicine: A Christian Approach to Medical Practice", edited by Vincent Edmunds, M.D., M.R.C.P., and C. Gordon Scorer, M.B.E., M.D., F.R.C.S.; 1958. London: The Tyndale Press. 8¼″ × 5″, pp. 192. Price: 12s. 6d. (English).

A discussion of medical ethics and conduct from a Christian point of view.

"Forensic Medicine", by Keith Simpson, M.D.; Third Edition; 1958. London: Edward Arnold (Publishers) Limited. 8½″ × 5″, pp. 360, with 137 illustrations. Price: 30s. (English).

The book has been extensively revised since publication of the second edition in 1952.

"Antibiotics Annual, 1957-1958", edited by Henry Welch, Ph.D., and Félix Martí-Ibáñez, M.D.; 1958. New York: Medical Encyclopedia, Inc. Distributed outside U.S.A. by Interscience Publishers, Inc., New York and London. 10″ × 6½″, pp. 1088, with many illustrations. Price: $12.00.

The proceedings of the fifth annual symposium on antibiotics held in Washington in October, 1957.

"Polysaccharides in Biology: Transactions of the Second Conference, April 25, 26 and 27, 1956, Princeton, N.J.", edited by Georg F. Springer, M.D.; 1957. Sponsored by The Josiah Macy, Jr. Foundation, New York. 9″ × 5¾″, pp. 248, with illustrations. Price: $5.00.

Contains verbatim accounts of group discussions on sialic acid and related compounds, on the interaction of polysaccharides and viruses and on pyrogens.

"Group Processes: Transactions of the Third Conference, October 7, 8, 9 and 10, 1956, Princeton, N.J.", edited by Bertram Schaffner, M.D.; 1957. Sponsored by The Josiah Macy, Jr. Foundation, New York. 9″ × 5¾″, pp. 328. Price: $4.00.

Contains verbatim accounts of group discussions on four subjects: interpersonal influences within the family, interpersonal persuasion, further studies on maternal-neonate interrelationships, Chinese Communist thought reform.

The Medical Journal of Australia

SATURDAY, MAY 31, 1958.

THE MEDICAL SCHOOL OF THE UNIVERSITY OF WESTERN AUSTRALIA.

THE formal opening of the new School of Medicine of the University of Western Australia, which took place on April 10, 1958, was an important and significant happening. The ceremony itself was simple. In the presence of the Premier of the State, the Lord Mayor of Perth, the Chancellor and other dignitaries of the University and about one thousand people, and after the appropriate speeches had been made, the State Governor, Lieutenant-General Sir Charles Gairdner, turned a golden key in the front door of the new School of Medicine building, which adjoins the Royal Perth Hospital. Just within the entrance door a bronze plaque has been placed which reads:

> The opening of this building on 10th April, 1958, by His Excellency the Governor, Lieutenant-General Sir Charles Gairdner, marked the establishment of the Faculty of Medicine of the University of Western Australia. This plaque also records the gratitude of the University to the Government and to the people of this State, whose generosity made possible the creation of a School of Medicine in Western Australia.

Thus, as the Dean of the new school, Professor Gordon King, has expressed it, the hopes and plans of many years reached fruition, and the ninth and youngest faculty of the University of Western Australia came into being.

The story of this new school will be familiar to readers of this Journal. For the past ten years and more it has been followed in detail.[1] A few only of the milestones need be mentioned now. In the early 1930's it was discussed by the medical profession, and certain preliminary steps were taken in relation to the study of anatomy. In 1946 Professor Peter MacCallum prepared and presented a comprehensive report to the Premier of Western Australia at the latter's request. In 1954 a series of circumstances brought the question again to the fore and made apparent to those in authority what had been apparent to others for many years—that a real and urgent need existed for Western Australia to train its own doctors. Early in 1955 a deputation from the Western Australian Branch of the British Medical Association waited upon the Premier to stress once again that no further time should be allowed to pass before a Medical School was established. The Premier promised that deputation that, if sufficient funds could be raised by public appeal, he would establish the school. He would set up a committee to advise the Government on all details concerning the proposed school. Since government support for the new venture was to be dependent upon a tangible expression of public opinion, a public appeal for funds for the new school of medicine was launched. A target of £400,000 was set, but in a remarkably short space of time a sum of well over half a million pounds was subscribed—the equivalent of approximately one pound per head from every man, woman and child in the State. Members of the medical profession alone contributed £40,000—the equivalent of over £70 per head of the medical population of the State.

Plans then proceeded apace. Provision was made for the erection of buildings for the teaching of anatomy, physiology and biochemistry in the fine and extensive university grounds on the Swan River at Crawley. Four of the main metropolitan hospitals were designated teaching hospitals—namely, the Royal Perth Hospital, the Princess Margaret Hospital for Children, the King Edward Memorial Hospital for Women and the Fremantle Hospital. It was decided that the already existing radium block in Victoria Square, next to the Royal Perth Hospital, should become the nucleus of the new school of medicine. After remodelling and the addition of a further storey, this building now houses the administrative offices of the faculty, common rooms and laboratories, two lecture theatres and the departments of pathology and microbiology. An excellent medical library exists in an adjacent building. This library was formerly the British Medical Association library, but the Association generously united with the Medical Faculty and the Royal Perth Hospital to set up the Medical Library of Western Australia, thus providing an invaluable collection of books and files of journals as the basis of a future library.

It was planned that teaching should commence at the beginning of 1957, and most of the senior members of the teaching staff were therefore appointed before the end of 1956. Teaching began simultaneously of first and final year classes in 1957, with 85 first-year students and 16 final-year students, all Western Australians who had returned to Perth from the University of Adelaide. In the current year four classes are under instruction, and by the beginning of 1959 all six years of the course will be running. Adelaide degrees are being awarded to students graduating in 1957 and 1958, but from 1959 onwards Western Australian degrees will be awarded to all new graduates. The provision of a curriculum has provided a remarkable opportunity to the teaching staff, especially as the General Medical Council in its latest series of "Recommendations" on the medical curriculum has provided for a much greater degree of latitude than in the past. The teaching staff has seized this opportunity and has exercised commendable imagination in its planning. The progress of the school will therefore be watched with the greatest interest by the medical profession throughout Australia and elsewhere. The only jarring note is the disappointment felt by some medical men in Western Australia that the organized profession was not represented or recognized at the formal opening ceremony. This feeling we share. It is appreciated that the University made clear their policy of issuing no personal invitations, as contributions to the new school had come from far and near. A general invitation was issued to all to attend. This was right, but it would surely not have cut across this policy to invite official representatives of the Western Australian Branch of the British Medical Association, the body which has the most direct interest in the school of

[1] M. J. AUSTRALIA, 1947, 1: 81 (January 18); 1947, 2: 145 (August 2); 1954, 1: 987, 994 (June 26); 1955, 1: 925 (June 18); 1957, 1: 252 (February 23).

any section of the community, which sent a deputation in 1955 to the Premier and so precipitated effective action, and which subscribed to the appeal with great generosity. This is not a carping criticism, and we sincerely hope that it will not be taken in that way. It is an expression of genuine regret. The members of the medical profession in Western Australia are interested in and proud of the new school. As we understand the position, they are only too willing to help it in any way in their power, but would not like to feel that they were not wanted. We are confident that they will join with this Journal in wishing our youngest medical school the fullest achievement of all its hopes.

THE MENACE OF SHOE-STORE FLUOROSCOPY.

THE present use of fluoroscopy during the fitting of shoes in shoe stores in New South Wales is farcical and dangerous. This is demonstrated beyond question by the thorough study by A. Bell and H. M. Whaite published in this issue. We have no details of the position in other States of the Commonwealth, apart from Victoria, which has attempted to regulate the practice; but there seems no reason to expect that, lacking effective controls, it is any better. The use of these machines should be forbidden by law, not only because of what they add to the radiation hazard, but also because of the pseudo-scientific humbug involved. The vast majority of customers and sales assistants can have little or no understanding of the X-ray shadows that they see, and it is painfully apparent that little is done to instruct sales assistants in this or in the reasonably safe use of the machines. It is significant to note that many stores have given up using them or have adopted subterfuges to discourage their use without offending their customers. If customer-pressure is the factor in perpetuating their use, abolition by law would solve the problem with no loss to anyone, and responsible proprietors of stores would heave sighs of relief. It is to be hoped that the N.S.W. State Health Department will heed this important study by its own officers and press on the State Government the need for firm, appropriate legislation, and that the Government will act promptly. Similar action, where needed, throughout Australia would be to the benefit of everybody.

Current Comment.

ENTERTAINMENTS IN HOSPITAL.

A good deal of publicity has been given to a recent decision of the Repatriation Commission to discontinue the holding of live-artist shows on Sunday nights at the Repatriation General Hospital, Concord, in New South Wales. A variety of people have expressed their views on the matter in public, and it has become something of a political football. One aspect of the subject about which not a great deal has been said, but which will interest our readers, is the medical viewpoint. This is well set out in a report submitted to the Deputy Commissioner for Repatriation by the Medical Superintendent of the Repatriation General Hospital, Concord, Dr. J. P. Taylor. This was read by the Minister for Repatriation, Senator Cooper, in reply to a question asked in the Senate on April 29, 1958. The reply is reported in *Hansard* for that date. In his report Dr. Taylor said that it was his opinion that in the general hospital the emphasis should be on treatment rather than on entertainment, and he felt that the live-artist shows on Sunday nights provided no real therapeutic benefit for the vast majority of the patients in the hospital. The bulk of the patients treated at the hospital were short-stay patients—the average over-all length of stay at present being around three weeks. The short-stay patient usually spent the greater part of his time while in hospital confined to his bed. At that time he was unable to attend such functions, and in any case it was not considered the necessary or proper function of a hospital to provide elaborate and highly organized entertainments, such as the live-artist shows, for that type of patient. Dr. Taylor went on to say that for the long-stay patient there was every justification for the provision of entertainment within the hospital. However, live-artist shows did not fill that need, for the following reasons. Long-stay patients might be divided into two sections: those suffering from tuberculosis, who comprise the majority of long-stay patients; those suffering from conditions other than tuberculosis, of whom there were sixty who had been in-patients for three months or longer. The second section might be further subdivided into (i) those with severe or terminal illness who were confined to bed, (ii) those with senile or arteriosclerotic mental deterioration, and (iii) a relatively small number, a dozen or so, with chronic disablement, such as paraplegia or joint or nervous system disease, who retained some mobility but remained in hospital because they either had no home or had unsuitable home conditions. For obvious reasons, those suffering from tuberculosis could not attend shows in a central hall. Of the second group, only those in sub-group (iii) could benefit. Apart from them the only persons likely to attend the shows were patients about to go home, resident hospital staff, patients' visitors and relatives, and, at times, strangers.

Dr. Taylor pointed out that the provision of over-generous entertainment and amenities at the Repatriation General Hospital aggravated the very problem constantly faced by the administration, of the existence of a substantial minority of "regular" patients, who, whilst not in need of in-patient treatment, constantly sought readmission because the conditions made hospitalization attractive to that particular type of patient. Moreover, apart from the lack of benefit derived from the live-artist shows, there was active interference with the well-being of sick patients, due to the late and noisy return to the wards on Sunday nights of patients who had attended the shows. Discussion with the matron and senior sisters confirmed the fact that great inconvenience was caused to the ward nursing staff by the functions. Dr. Taylor said that he was convinced that the entertainment needs of patients at the hospital were adequately provided for, apart altogether from Sunday night live-artist shows, by the entertainment and amenities otherwise available. He then set out a quite considerable list of the entertainments available, and concluded by saying that he considered the discontinuance of live-artist shows fully justified on the following grounds: (*a*) that no real need existed for them, (*b*) that they interfered with the welfare of sick patients and caused unnecessary inconvenience to the nursing staff, and (*c*) that they aggravated certain administrative problems.

Senator Cooper referred also to a report from the senior medical officer of the Repatriation Department in New South Wales, who expressed complete agreement with Dr. Taylor's views, and pointed out that what had happened had not been a sudden and isolated decision, but was part of a line of action over the last five or six years designed "to concentrate the activities of the institution on those things in hospital life which really aid the sick, whether they be 'straight' medical and surgical treatment, or ancillary entertainments and amenities". The Deputy Commissioner for Repatriation and the Minister for Repatriation himself endorsed these views.

To medical readers, little further comment is required. The primary object of any hospital is to see to the treatment of sick patients. Any considerations which cut across that object must be regarded as secondary and be dealt

with accordingly. In most hospitals no one would think of disputing this, but in such places as repatriation hospitals sentimental and other influences can confuse the issue. There seems little doubt that in the present instance a wise decision was made.

POLIOMYELITIS.

FOUR CONCLUSIONS of a World Health Organization study group may fairly be regarded as summing up the public health position of poliomyelitis on a world level: the results of vaccination campaigns against poliomyelitis justify the use of vaccination in countries where poliomyelitis is an important problem; live poliovirus vaccines are still experimental and cannot yet be used in public health programmes; in countries that have reported few cases of paralytic poliomyelitis, the actual poliomyelitis problem may be as much as ten times larger than available figures indicate; all countries should have laboratory services for diagnosing virus diseases. These are some of the conclusions reached by a Study Group on the Control of Neurotropic Virus Diseases, which met in Copenhagen from April 14 to 19, 1958, at the Regional Office for Europe of the World Health Organization.

It appears that virus diseases affecting the nervous system are a growing problem in the European region. Particularly for poliomyelitis, more information on the occurrence of the disease is urgently needed, but in many countries reliable data are not available and underreporting is general. In order to obtain a true picture of the importance of poliomyelitis it is necessary also to consider the accumulation of paralytic cases. Each year a new group of cases of permanent paralysis is added to the large numbers remaining from earlier years. The study group recommended that studies be made in order to assess the true medical, social and economic consequences of poliomyelitis.

On the question of vaccination, it is pointed out that epidemic waves of poliomyelitis have been occurring every few years and, because of this periodicity, it is too early to be quite certain about the value of mass vaccination campaigns against poliomyelitis. Yet the incidence of paralysis has gone down after such campaigns, and these apparent results are sufficient to justify the continuation of campaigns or the starting of new campaigns in countries where poliomyelitis is important. However, in some countries insufficient information is available, and more precise data should be obtained before an expensive vaccination programme is started. It will also be necessary to consider the results likely to be obtained in relation to the other demands on the limited resources available for health programmes. Accidents following the use of poliomyelitis vaccination have been very few because of the great care that has been taken during vaccine production and tests.

With regard to the much discussed question of live poliovirus vaccines, the view held is that, although results are in part very promising, these vaccines are still experimental. The group could not forecast when enough information would be available to justify their large-scale application. In the meantime, the group recommends caution in their experimental use.

A major recommendation made was that virus diagnostic laboratory services should be made available in all countries for the following reasons: (i) to inform the public health authorities of disease-causing viruses existing in the country, and so enable them to take appropriate preventive action; (ii) to assist in carrying out epidemiological investigations and surveys, and to improve the reporting of communicable diseases; (iii) to evaluate control measures such as vaccination; (iv) to ensure the proper approach to prevention and the early treatment of disease. Though specific laboratory diagnosis of most virus diseases may be of little value for individual cases, an efficient laboratory service may be able to provide very useful pointers to the doctor in charge of the patient.

MEDICAL SCHOOLS AND DOCTORS OF THE WORLD.

AUSTRALIA is much interested in medical schools just now, but our needs and problems look small when we set them in the world picture, as it is shown in the new "World Directory of Medical Schools" published by the World Health Organization.[1] According to this authority, 108 new medical schools have been created in the past 10 years, more than at any other time in history. There are now 638 medical schools in the world, situated in 84 countries and territories; of these 10 were founded before the year 1300. From them all more than 66,700 new doctors graduate each year, to join their older colleagues, whose number throughout the world is estimated to be about 1,200,000. In most countries, men and women students are admitted to medical studies on the same basis; in at least one country, Afghanistan, male students only are eligible. As a rule, the number of men students is far in excess of the number of women students; but there are a few exceptions, notably the U.S.S.R., where women students often are two or three times more numerous than men.

One fact startlingly apparent from the Directory is the unequal distribution of medical schools and of doctors throughout the world. The following figures bear this out. In Africa, with a population of 210,832,000, there are only 16 medical schools, which produce 1089 graduates each year (one per 190,000 of population); the total number of doctors is 23,284 (one per 9000 of population). In North and Central America, with a comparable population (234,276,000), there are 124 medical schools, 9076 graduates each year (one per 26,000 of population) and a total of 259,664 doctors (one per 900 of population). In South America the population is 121,000,000, the 53 medical schools produce 5308 graduates annually (one per 23,000), and the doctors total 48,263 (one per 2500). In Eastern Asia, with a population of 1,370,821,000, there are 171 medical schools, 12,418 graduates (one per 110,000) and 209,688 doctors (one per 6500). In Western Asia, with a population of 82,529,000, there are 15 medical schools, 868 graduates (one per 95,000) and 16,951 doctors (one per 4900). Europe, with its population of 619,707,000, has 253 medical schools, which pour out 37,222 graduates (one per 17,000) each year to swell the total of 665,522 doctors (one per 930). In Oceania, the population is 14,234,000, the medical schools number only six but produce 741 graduates (one per 19,000) and the total number of doctors is 12,427 (one per 1150); as this area includes Australia, New Zealand and Hawaii, the figures for which are much more favourable than those for the whole area, it is apparent that in Oceania too there is an unequal distribution of medical care. The picture is completed with the total world figures: the population is 2,653,399,000—just a little less than twice the total for Eastern Asia; the number of medical schools totals 638, and they produce 66,722 new graduates (one per 40,000) each year; the doctors number 1,235,799 (one per 2150 of population). Thus the ratio of doctors to population ranges from one in 900 in North and Central America, through decreasingly favourable figures in Europe, Oceania, South America, Western Asia and Eastern Asia to Africa's depressingly low score of one in 9000; and it has to be borne in mind that some individual countries, particularly in Eastern Asia, have worse figures than this—the worst is Nepal and Bhutan (one in 174,640). The ratio of new graduates produced annually to total population is highest in Europe (about one in 17,000), decreasing steadily through Oceania, South America, North and Central America, Western Asia and Eastern Asia to Africa's figure of one in 190,000.

A great deal of other information is available from this Directory, which deals with the medical schools of each country in turn, setting out details of such things as administration, conditions of admission, curriculum, examinations held and qualification requirements. It is a most useful and interesting publication for all who are concerned with medical education and its academic and social implications.

[1] "World Directory of Medical Schools"; 1957. Geneva: World Health Organization. 9¼" × 6¼", pp. 314. Price (cloth-bound edition): £1 5s., $5.00, Sw. fr. 15.

Abstracts from Medical Literature.

MEDICINE.

Pulmonary Embolism.

H. L. ISRAEL AND G. GOLDSTEIN (*Ann. Int. Med.*, August, 1957) discuss the varied clinical manifestations of pulmonary embolism. They state that the condition is a common finding at autopsy, although regarded in many hospitals as an unusual disorder, and infrequently considered in the differential diagnosis of cardio-pulmonary diseases. The frequent failure to recognize pulmonary embolism clinically results from widely held misconceptions regarding the prevalence of this disorder, and the evidence necessary for its diagnosis. If the diagnosis is considered only when all the classical clinical and laboratory features are present, most instances of embolism will go unrecognized. Where physicians are familiar with the variety of clinical, radiological and electrocardiographic manifestations which characterize pulmonary embolism, the frequency and importance of this disorder in modern practice become evident. The authors present their observations over an eighteen-month period at a large general hospital. Ninety instances were recognized in this particular study, outnumbering pneumonia and bronchogenic carcinoma. Embolism was preceded by surgery in 33 patients, and by musculoskeletal trauma in six patients. It occurred in 18 patients admitted to hospital with medical illnesses. In 33 patients embolism occurred before admission to hospital. Respiratory symptoms predominated in 39 patients and cardiovascular symptoms in 33 patients; abdominal symptoms were most prominent in six cases, and central nervous system manifestations in four cases. The disease requiring differentiation from embolism most often was acute myocardial infarction. Chest skiagrams are important in the diagnosis of pulmonary embolism, and although there is no pathognomonic configuration, this possibility was suspected in 55% of patients with pulmonary embolism who were examined by X rays. Electrocardiographic abnormalities were detected in 70% of cases adequately examined. Typical *cor pulmonale* patterns were observed in seven patients, transient positional changes in 28, and coronary insufficiency patterns in 18 cases. The high frequency of transient electrocardiographic changes observed was remarkable, since the episodes of embolism were so often of moderate or slight severity. Serum glutamic-oxalacetic-transaminase determinations were made in 49 patients, and levels were consistently normal in 32 patients while elevations were noted in 17 patients. In all but one of these associated hepatic, myocardial or musculoskeletal diseases accounted for the elevation. In the absence of these complicating factors, transaminase determinations were valuable in the differentiation of pulmonary and myocardial infarction. The authors conclude that pulmonary embolism will more commonly be recognized when it is appreciated as being the most common lung disease now encountered in general hospitals, when this diagnosis is given first consideration in a wide variety of syndromes, and when informed use is made of chest skiagrams, electrocardiograms and serum transaminase determinations.

Febrile Transfusion Reactions.

T. E. BRITTINGHAM AND H. CHAPLIN, JUNIOR (*J.A.M.A.*, October 19, 1957) discuss febrile transfusion reactions caused by sensitivity to donor leucocytes and platelets. Unexplained febrile reactions to transfused blood are frequent, especially in patients who receive multiple blood transfusions. These reactions have been regarded as non-specific when there is no evidence of blood contamination, pyrogens, red cell incompatibility or sensitivity to the donor's plasma. The authors report the presence of strong leuco-agglutinins in the sera of most patients who had received repeated transfusions and had had severe febrile reactions. The authors tested 10 patients who needed transfusions. Five patients (group A) had previously had from 20 to 85 transfusions with some febrile reactions. They had demonstrable antibodies against the normal leucocytes given during this study. The other five patients (group B) had had only seven or less transfusions and no reactions. Group A did not develop reactions when the blood used for transfusion was prepared by centrifuging to remove as much as possible of the buffy layer, but all reacted with fever and other symptoms, and gave typical laboratory findings when transfused with the buffy coat which had been removed. Group B did not react adversely to either fraction of the donors' blood. Evidence suggested that it was the leucocytes rather than the platelets in the buffy layer which caused the reactions. It was suggested that techniques for removing the buffy coat when blood is prepared for transfusion may prevent dangerous reactions, when patients become isoimmunized to donor leucocytes after repeated transfusions.

Streptococcal Infections.

A. J. MORRIS AND C. H. RAMMELKAMP (*J.A.M.A.*, October 12, 1957) record the results of an investigation to determine the effectiveness of benzathine penicillin G in preventing streptococcal infections of the throat. An epidemic of pharyngitis due to group A streptococci occurred among airmen at Warren Air Force Base, Wyoming. Sixteen per 1000 airmen were affected. At this stage benzathine penicillin G was given to 10% of the men. Three different doses were used: 300,000, 600,000 and 1,200,000 units. Two hundred and fifty airmen were given 300,000 units, and 264 men in the same group were given saline solution only, as controls. In another group 250 men were given 600,000 units, in a third group 250 men were given 1,200,000, and in each group an equivalent number of men were given saline injections. In the first week after the injections no streptococcal illness was noted in the penicillin groups, and 26 cases in the control groups. In the second week two infections occurred in the group of men who had received 300,000 units and none in those who had received the larger doses. Oropharyngeal swabbings were taken daily, and it was shown that, among the men who were given penicillin, no sore throats occurred until three to five weeks later among those who had received 600,000 units, and six to seven weeks later among those who had received 1,200,000 units. The carrier rate in those who had received 300,000 units was reduced to one-tenth of that in the control groups, among whom 80% had group A streptococci in the throat during the follow-up period. The authors conclude that benzathine penicillin G in doses of 300,000 up to 1,200,000 eradicated streptococci from most carriers, and that the larger dose gave the longest protection, lasting six to seven weeks.

A Test for Atheroma.

E. M. M. BESTERMAN (*Brit. Heart J.*, October, 1957), in a study of the serum lipoproteins by paper electrophoresis, sets out to evaluate the relationship of a "pre-β"-lipoprotein to atheroma and to coronary arterial disease in particular. The pre-β-lipoprotein had first been noted by Dangerfield in 1955, and was separated by electrophoresis in a band between the α- and β-lipoproteins. It was now found in 198 of 200 patients with ischæmic heart disease and in 42 patients with dominant aortic atheroma, and it occurred to a lesser extent in some of the 124 control subjects. Statistical comparison between the lipoprotein fractions in the sera of ischæmic and control subjects showed a consistently significant difference only in the pre-β-lipid.

Cholesteræmia and Nicotinic Acid.

P. O. O'REILLY, M. DEMAY AND K. KOWLOWSKI (*Arch. Int. Med.*, November, 1957) discuss cholesteræmia and nicotinic acid. They state that it is generally accepted that the development of arteriosclerosis is accompanied by, if not due to, a disturbance in the metabolism of lipids. Total cholesterol, cholesterol esters, fatty acids, phospholipids and lipoproteins have been incriminated singly or in the ratios or combinations of these variables. There is experimental, clinical and histopathological evidence that a disturbance in cholesterol metabolism is involved in the development of arteriosclerosis. Although hypercholesteræmia does not necessarily assume a major role in the genesis of arteriosclerosis, it may be indicative of developing atherosclerosis, though the converse does not necessarily hold. Many approaches have been made to the problem of reducing the cholesterol level in the blood. These include dietary restrictions, hormonal therapy, vitamin *B* complex, and colloid stabilizers such as lecithin and albumin. Recent investigations with the administration of nicotinic acid to rabbits and humans stimulated the investigations undertaken by the authors. Forty-five patients and two volunteers were studied, and the fasting total serum cholesterol was determined for each subject. Some were given one gramme of nicotinic acid by mouth three times daily for three months, while others received no nicotinic acid therapy. The serum cholesterol levels were redetermined at intervals of one week and six weeks after

the beginning of the experiment. At the end of three months additional estimations of serum cholesterol were made on those receiving the nicotinic acid therapy. The author states that this study appears to confirm two facts: (*a*) that nicotinic acid does lower the serum cholesterol level, and (*b*) that its lowering action appears to be proportionate to the level of blood cholesterol. They proceed to discuss the mode of action of nicotinic acid, and state that the protective activity of the nicotinic acid resembles the protective activity of lipotropic factors. When given to patients with initial low serum cholesterol, nicotinic acid tends to increase its level, whereas in cases with high serum cholesterol level it tends rapidly to reduce it to a normal level. They discuss the relation between nicotinic acid and the anti-lipæmic action of heparin.

Dyspepsia After Coffee.

P. BRUMMER AND E. SYSIMETSÄ (*Acta med. scandinav.*, volume 159, fasciculus 2, 1957) observed by fluoroscopy the gastric motility in test subjects after the ingestion of black coffee and of coffee with cream. There were 11 subjects who regularly suffered dyspeptic distress after coffee with cream and 11 who did not. In those who had no symptoms it was found that there was no difference in gastric motility whether the coffee was taken with cream or not. Persons who had gastric distress after coffee with cream showed immediately after ingestion a decrease in peristalsis and delayed gastric emptying as compared with the sequence of events after taking black coffee.

Salt Intake and Hair Loss.

E. FOLDES (*Acta med. scandinav.*, volume 159, fasciculus 2, 1957) counted the number of hairs which came out of the scalp each day under experimental conditions in a man who was becoming bald on top. He found that the daily loss of hair was fairly constant; but adherence to a salt-poor diet significantly reduced the loss of hair, while after two days or more of unrestricted salt intake the rate of loss of hair was always significantly increased.

Penicillin Prophylaxis.

R. W. SHERWOOD *et alii* (*J.A.M.A.*, October 12, 1957) report on the effects of multiple injections of benzanthine penicillin G in a United States Army unit as a protection against streptococcal infections, rheumatic fever and nephritis. One group of 1567 men received 600,000 units every four weeks, and another group of 1708 men received 900,000 units every six weeks. No anaphylactoid reactions occurred, but during six months 16 men were admitted to hospital with urticaria, angioneurotic œdema or joint symptoms; 11 men were admitted after the first injection, four after the second, and one after the third injection, the likelihood of a reaction decreasing with subsequent doses. Those men who developed reactions received no further injections of penicillin. Prolonging the interval between doses did not affect the likelihood of a reaction. Men who had previously suffered from skin rashes or joint swelling after penicillin injections were excluded from the trial. Streptococcal infections were rare, but gonococcal infections occurred in one of 3275 treated men as against 192 of the 13,000 men who had received no injections. The authors conclude that benzathine penicillin G is a safe prophylactic measure against streptococcal infections.

Chronic Intolerable Pain.

K. O. VON HAGEN (*J.A.M.A.*, October 19, 1957) discusses the mechanism of chronic intolerable pain and describes the successful use of electro-shock therapy in the cases of eight women between the ages of 45 and 62 years who complained of pain, generally of an unusual type, without obvious organic disease. The pains of which the patients complained included abdominal pain, thumping pain in the neck and head, pain following a cut finger, constant headache following hemiplegia, and pain in the legs following an accident. In all patients the pain was alleged to be intolerable, was unrelieved by drugs or surgery, and was associated with depression or an emotional state. The conditions appear to have been psychological. Electro-shock treatment was given as an alternative to frontal lobotomy. All the patients improved markedly. The treatment was often prolonged and several courses of electro-shock treatment were given, associated with drug treatment and supportive psychotherapy.

Perthane and Adreno-Cortical Function in Man.

I. TALIAFERRO AND L. LEONE (*New England J. Med.*, October 31, 1957) investigated the properties of perthane, an insecticide structurally related to DDT, which has the apparent property of causing adreno-cortical atrophy in the dog. Using human subjects with advanced malignant disease, and doses of 150 to 300 milligrammes per kilogram of body weight per day, depression of the response to ACTH stimulation was obtained, as measured by plasma levels and urinary output of 17-hydroxy-cortico-steroids.

Tolbutamide and Diabetes.

D. HURWITZ AND A. C. MCCUISTON (*New England J. Med.*, November 7, 1957) report the effects of tolbutamide, an oral hypoglycæmic agent, in the treatment of *diabetes mellitus*, and compare its effects over short periods with that of a placebo in a double blind trial on outpatients, previously taking insulin, using analysis of urine and fasting and postprandial blood sugar levels as criteria. Diabetic diets adjusted for the individual were also prescribed. Although 60% of patients had good control with diet and tolbutamide, no less than 22% had good control with diet and placebo.

Primary Varicella Pneumonia.

S. KRUGMAN, C. H. GOODRICH AND R. WARD (*New England J. Med.*, October 31, 1957), reporting from the Bellevue Medical Centre, New York City, describe 10 cases of varicella in adults with pulmonary involvement. No changes were seen in a larger number of children admitted with the same disease during the same period. Clinically the patients complained of cough and hyperpnœa without chest pain, commencing two to five days after the onset of the skin eruption. Half the patients had either cyanosis or hæmoptysis. Antibiotics did not appear to alter the course of the pulmonary involvement which subsided within three days, although pleural effusion was an occasional complication. Diffuse coalescent nodular shadows were visible on radiological examination in all cases. Leucocytosis was only occasionally present. One young adult died, and autopsy revealed many areas of lobular consolidation with fibrinous exudate containing mononuclear cells, some with intranuclear inclusion bodies, as the principal cell type.

Antibiotic Prophylaxis in Coma.

R. G. PETERSDORF *et alii* (*New England J. Med.*, November 21, 1957) report a study of 72 unconscious patients of middle age and beyond, who survived more than two days after admission at the Johns Hopkins Hospital. The majority were cases of cerebral vascular accident. The effects of antibiotics as prophylactic agents against infection were studied, using a control group of patients who did not receive antibiotics. There was no difference in the hospital mortality rates between the two groups. Pneumonia was less frequent at autopsy in the group who had not received prophylactic antibiotics. Chemoprophylaxis did not prevent urinary tract infection after single catheterization, and where indwelling catheters were used more organisms of other species refractory to antimicrobial therapy appeared in the treated group. Two deaths with Gram-negative bacteriæmia occurred, both in the treated group. It is concluded that antibiotic prophylaxis in unconscious patients is of no benefit and is distinctly hazardous.

Rheumatoid Arthritis.

A. COHEN *et alii* (*J.A.M.A.*, September 21, 1957), who evaluated treatment of rheumatoid arthritis with prednisone for more than a year, were left with little doubt that prednisone and prednisolone are superior to cortisone, hydrocortisone and corticotrophin. The incidence of duodenal ulcer amongst their patients under treatment was no greater than in the general population. Treatment was started with a dosage of 20 milligrammes daily, reduced to 17·5 milligrammes when improvement was apparent. Reduction in dosage by decrements of 2·5 milligrammes weekly was continued so long as no symptoms returned.

Pancreatic Lesions in Patients Treated with Corticoids.

F. A. CARONE AND A. A. LIEBOW (*New England J. Med.*, October 10, 1957), reporting from Yale, studied the post-mortem incidence of pancreatic changes in patients treated with ACTH or adreno-cortical steroids. In 16 out of 54 such patients there was histological evidence of acute interstitial pancreatitis or fat necrosis, or both, not explicable by the primary disease, and in only one out of 54 control cases. No clinical counterpart to the histological findings was evident. Acinar ectasia was also found in 59% of the treated group and in 24% of the control group. There were no cases of acute hæmorrhagic pancreatitis.

On The Periphery.

WHY NOT MOTOR?

> The use of travelling is to regulate imagination by reality, and instead of thinking how things may be, to see them as they are.
>
> —DR. SAMUEL JOHNSON.

GOING abroad may involve travel by land, by sea or by air. All three methods of travel to and from the United Kingdom are not always considered or even thought feasible. It is my intention to introduce the perhaps novel idea of driving to London. Naturally one must ship oneself and a vehicle, unless one hitch-hikes (a fascinatingly uncomfortable possibility) to either Africa or India. It is possible, but not easily practicable, to motor up the Malay Peninsula through Burma to India.

The main necessities are a car, time to spare (three months via India and four via Africa) and a spirit of adventure. If one does not include loss of income, then the cost of two people motoring to India or Africa from the United Kingdom is very little more than the cost of shipping the car and themselves direct to Australia, supposing they camp wherever possible and avoid expensive hotels. Any vehicle in good mechanical condition with a good set of tires is suitable, particularly on the Indian trip. My wife and I drove a cheap 10 horsepower car to Bombay from London in 1955 with little more trouble than a couple of broken springs and two punctures. Except in the Sahara desert, petrol is plentiful and cheap, and no special provision for carrying petrol need be made beyond carrying a few extra gallons in tins over the long stretches. Crossing the Nubian desert—an offshoot of the Libyan desert—in 1953 in a large station wagon, we used 34 gallons to cover 250 miles, but this is exceptional, and before such "horror" stretches petrol in sealed four-gallon tins is usually available.

Via India.

The terminal points for this route are one of the main seaports in India—Bombay, Calcutta or Colombo. All routes lead through Delhi into West Pakistan and lead either directly into Iran (Persia) or north through the Khyber Pass into Afghanistan. Visas for Afghanistan are easy for *bona fide* medical practitioners to obtain, but any mention of literary or journalistic interests would probably prevent entry. The mountains in Afghanistan and parts of Iran must be traversed before winter, when snow blocks the passes and streams are unfordable. These countries should be crossed before December; spring and autumn are the best times of the year to travel, when the long hot desert stretch in Iraq and Jordan is comfortably crossed. From Jordan and Syria the route passes across Turkey to enter Europe at Istanbul. This route is good, with rough roads east of Baghdad. There are almost no bridges east of Teheran, but streams are easily forded except after heavy rain. From whichever port one takes a ship in India, a large part of the country with all its fascinating colourful civilizations is easily visited. Afghanistan is a wild and rugged primitive country largely populated by nomads. Persia, and more particularly Iraq, have remnants of the very early civilizations which can be seen. Babylon and the Tower of Babel are not far from Baghdad. To mention other places *en route* is merely to recite a list of most places mentioned in the itinerary of any Middle East guide books, such as Teheran, Baghdad, Jerusalem, Damascus, Beirut, Aleppo, Ankara and Istanbul.

Via Africa.

This road is longer and more arduous than the Indian and Asian route. From Cape Town the road goes north through Rhodesia. One may then keep to the right and follow the Cape to Cairo route, or bear left through the Congo and across the Sahara to the North African coast. Both routes are impassable at various times of the year. The Sahara is "shut" from June till November because of heat, when even the famous French trans-Sahara transports do not run. This road, whilst it crosses 2000 miles of desert, is fairly easily passed. It is said that everyone gets bogged once and then learns the niceties of driving in sand and has no further trouble. The Cape to Cairo route leads through British East Africa (Tanganyika, Kenya and Uganda) into the Sudan. The track first follows the White Nile through the Sudd, that vast papyrus-covered area which is swamped by the overflowing Nile about July each year, causing all wheeled transport to bog for about three months. From the North African coast it is not possible to drive to Europe, for the present political situation makes it impossible to drive out of Egypt into the Middle East. The Egyptian-Israeli border is closed to all travellers. It is convenient and comparatively cheap (Mediterranean shipping charges are fantastic) to travel by ship from Alexandria to Beirut.

The African route is longer and more rugged than the Indian one, but it offers a greater variety of interest. Wild game of all kinds is plentiful and easily seen throughout the country. Ancient Egyptian remains at Luxor and Thebes fascinate even the most disinterested student of ancient history. Native and "colour" problems are rife and they vary throughout the country. Perhaps one should not forget to mention the Victoria Falls at Livingstone.

Political situations vary from day to day, but never seem as bad when one is on the spot as the newspapers would have one believe. Australian travellers appear to be universally popular, except possibly in France, provided they conveniently ignore their status as "Britishers" (the passport defines an Australian as "An Australian citizen by birth and a British subject"); local people are very friendly and helpful. My wife had her face gashed in north-west Afghanistan by a stone thrown by an incensed "mullah", for she had appeared in public without adequate face coverings—an offence to an ultra-religious and backward Mohammedan. But I have experienced no other unpleasantness from "locals" other than the boorishness one has to expect from Egyptians in their own country.

FIGURE I.
Major road routes.

All medical graduates at some time decide whether to stay in their own country or to go abroad for further post-graduate training. Having decided to go, they make an attempt, often abortive, to secure a passage as a ship's surgeon. Failing in that attempt, they meekly pay for a passage. For these people I present the overland routes as an alternative to conventional shipboard life.

> Slumber not in the tents of your fathers, the world is advancing, advance with it.—MAZZINI.

Mount Gambier, South Australia.

D. N. HAWKINS.

Clinico-Pathological Conferences.

A CONFERENCE AT SYDNEY HOSPITAL.

A CLINICO-PATHOLOGICAL conference was held at Sydney Hospital on August 20, 1957; DR. WILFRED EVANS, honorary consultant physician, was in the chair. The principal speaker was DR. JOHN READ, assistant honorary physician.

Clinical History.

The patient was a woman, aged 67 years, whose illness had commenced four days before her admission to hospital, with anorexia and cough. Soon afterwards she had cold shivers, and

the cough became very much worse and productive of blood-tinged sputum. On the day before her admission she vomited some "coffee grounds" material, and later that day she coughed up some blood. She developed chills again and pains in the chest, back and legs. In the early hours of the following morning she lapsed into stupor. No details of her past health or family history were available.

Examination of the patient revealed her to be semi-conscious—she reacted to painful stimuli, but did not respond to the spoken word. She was restless and moaned intermittently. Her temperature was 101·4° F. Her pulse rate was 120 per minute, with an occasional missed beat and sometimes coupled beats. The position of the apex was not recorded. The heart sounds were normal. The respiratory rate was 35 per minute and the respirations were grunting. There was dullness to percussion at both lung bases, more marked on the right side. The vocal resonance and fremitus and the breath sounds were regarded as within normal limits. Bronchial breathing was absent. The tongue was dry, and was coated with dark dried blood. Tenderness and guarding were present in the epigastrium, but there was no rigidity and the abdomen moved on respiration. There was no abdominal distension. The patient was staring into space. The eyes were making conjugate movements. Marked neck rigidity and Kernig's sign were present. The reflexes were generally increased and equal. The plantar responses were flexor. The pupils and cranial nerves were normal. There was no paralysis. Tenderness was noted at both renal angles. The specific gravity of the urine was 1·020 and albumin and a heavy deposit of acetone were present, but there was no reducing substance. A lumbar puncture was performed. The cerebro-spinal fluid was xanthochromic and cloudy, and the pressure was 170 millimetres of cerebro-spinal fluid. The cell count was 350 erythrocytes and 48 leucocytes per cubic millimetre. The chemical findings were as follows: sodium chloride content, 600 milligrammes per 100 millilitres; glucose content, 48 milligrammes per 100 millilitres; protein content, 1000 milligrammes per 100 millilitres. A blood count gave the following information: the hæmoglobin value was 15 grammes *per centum*; the leucocytes numbered 15,000 per cubic millimetre, 74% being neutrophils (which showed a "shift to the left"), 22% lymphocytes, 3% monocytes and 1% eosinophils.

Penicillin was administered intrathecally and intramuscularly, "Trisulpha" and "Aureomycin" were given by mouth, and symptomatic treatment was given. The urine was free of sugar for eight hours and the ketonuria diminished. At this time, when a trace of ketones remained, some reducing substances were found, and the amount of these increased to "++" four hours later. Shortly afterwards the patient died.

Clinical Discussion.

DR. JOHN READ: It is customary at many of these exercises for the speaker to go through the history again, a most boring procedure when you have it in front of you. So I briefly present my version of the summary which you see on the blackboard: we are dealing with the problem of an elderly woman who had a disease of apparently acute onset and rapidly fatal termination. She had a number of symptoms and signs which we may group as follows. She had chills and cold shivers and generalized aches and pains, accompanied by fever and a neutrophil leucocytosis. From these I think that we may infer that she had an infection.

She had cough and hæmoptysis, rapid grunting respiration, and dullness at her lung bases. From this I think that we may reasonably infer that she had respiratory disease.

It is said that she was vomiting coffee-grounds material and that she had anorexia. Her epigastrium was tender to palpation, she was dehydrated, and we may suggest that perhaps she had gastro-intestinal disease. The blood in her mouth could have come equally well from her lungs or stomach. She was stuporose and she had meningism, and an abnormal cerebro-spinal fluid. It goes without saying that she had meningeal disease.

Examination of her urine showed a fairly high specific gravity, albuminuria, acetonuria, and later in the course of the disease, reducing substances. Again, with queries in front of both, we have the possibilities of renal disease and of *diabetes mellitus*. There is nothing very profound in that summary. It merely defines the limits we may go to. We may decide to dismiss some of these things, but certainly outside those interpretations I feel we cannot go. We are specifically told, to complete the story, that there were no abnormalities of the breath sounds, no accompaniments, no cardiac murmurs and no focal neurological signs.

To expand a little on these interpretations before discussing specific diseases: The history, first of all, is obviously scanty. We are not told where it came from. In her mental state, it is presumed that it came from family or friends. We do not know whether she was living with them and whether she may have had symptoms for some time before this four-day period. Did she have earlier symptoms, and perhaps more important, did she have earlier treatment outside the hospital? That may well have modified the course of the disease under discussion, given for example that she had had some penicillin or broad-spectrum antibiotics.

So we shall not lean too heavily on the history if it should conflict with our other interpretations.

Under the heading of physical examination I shall make a number of complaints. I feel that I have been left rather short of information, not on particularly high-powered tests, but on things which might well have been mentioned in the physical examination. We are not told her blood pressure. We are told that she had albumin in her urine, but not how much albumin—whether it was a trace or "three-quarters". And we are not told the condition of the fundi, if they were inspected. Those three things would have helped.

As far as the specific features are concerned, infection I think is straightforward, with one reservation to be mentioned later. The respiratory disease we will talk a lot about later. It is interesting that she is recorded as having dullness at both bases, worse on the right side, yet her breath sounds were perfectly normal in quality and intensity and she had no adventitiæ. I know of no respiratory pathology which will produce this combination of signs in the chest, so we shall ignore for the remainder of this discussion the question of signs in the chest and assume that all or any may have been present or absent.

Is the gastro-intestinal tract at fault at all? We are told she had anorexia. This is quite non-specific. If she was sick she may well have had anorexia. We are told that she was vomiting coffee-grounds material. The description "coffee-grounds material" is, in my experience, a very loosely used term to signify anything dark that comes from the patient's mouth. In this case there are three possible interpretations, genuine gastro-intestinal bleeding, swallowed blood, or merely dark secretions in a woman who was vomiting. The question of tenderness is very hard to interpret, both in the epigastrium and in the renal angles. This woman is recorded as being stuporose, responding only to painful stimuli. We may either infer that the tenderness was very extreme, or just that she was fortuitously jumping around as the resident pushed her back and front.

As far as the central nervous system is concerned, we know that she had meningitis. That part is easy. The cerebro-spinal fluid was xanthochromic, which is, of course, not specific for previous bleeding into the subarachnoid space. It can as well be associated with a very high protein content or the presence of dead leucocytes. We are told that the fluid contained 350 red cells and 48 white cells. The pathologist did not tell us what the 48 white cells were. The cerebro-spinal fluid protein is very high, 1000 milligrammes per 100 millilitres, the glucose I would prefer to regard as within normal limits (and anyway it does not matter), and the chloride is pathologically low. The pathologically low chloride is not pathognomonic of certain forms of meningitis. It can as well be a reflection of the general state of body chloride, and I certainly have seen at various times people with Addison's disease or diabetic ketosis and vomiting who had cerebro-spinal fluid chlorides very much lower than this without nervous or meningeal disease. The pressure of 170 is equivocal. Certainly we are not going to say that it is high. In an uncooperative patient, the most that we can say is that it is not low. Another thing we are not told is the Queckenstedt response, which again would have helped.

The kidneys we might discuss a little later.

What are we to regard as the possible diagnoses in this case? We can say that the most likely group is that of an infective, central nervous system-respiratory disease. I feel that the diagnosis must really lie in this group.

The first one to be discussed will be tuberculous infection of lungs and meninges. We would then postulate pulmonary tuberculosis, tuberculous meningitis, and perhaps miliary tuberculosis. She had symptoms which are altogether compatible with pulmonary tuberculosis: cough, fever, hæmoptysis,

malaise. She may well have had chronic pulmonary tuberculosis with or without a recent acute spread. The coffee-grounds vomitus might well come from swallowed blood. We have seen patients with large cavities in their lungs due to tuberculosis, who are coughing up a little blood, vomiting quite a lot of altered blood, and at autopsy there is no lesion in the stomach to account for the bleeding. We can only assume that they are swallowing more blood than they are coughing up. So all this is compatible with pulmonary tuberculosis. What about tuberculous meningitis? We know that she had meningitis with a low cell count in the cerebro-spinal fluid. The cells in tuberculous meningitis may number anything from a few to over a thousand. The type does not matter a bit. They may be all polymorphs or all lymphocytes or any mixture of the two at any stage of the disease. More commonly they are predominantly polymorphs during the so-called intrathecal tuberculin reaction after treatment has commenced. But even in the untreated disease there may be spontaneous bursts of almost pure polymorphs. So at this point I do not particularly mind what the leucocytes were.

The rise of cerebro-spinal fluid protein is again classical of tuberculous meningitis, but in acute cases one would like it to be rather closer to 100 than to 1000 milligrammes per 100 millilitres. In the straightforward case of tuberculous meningitis, the level is usually around several hundred milligrammes per 100 millilitres. The level, of course, may be very high. I have seen one case where the cerebro-spinal fluid protein level reached 3·5 grammes per 100 millilitres, and a record of another case in which it reached 11 grammes per 100 millilitres. Such levels as these, or even one of 1000 milligrammes per 100 millilitres, are usually associated with loculation in the subarachnoid space and the presence of subarachnoid block. Indeed, in recent years some of us have regarded a high or a rising cerebro-spinal fluid protein in tuberculous meningitis as an indication for steroid therapy. So if this is tuberculous meningitis, a cerebro-spinal fluid protein of 1000 milligrammes per 100 millilitres suggests a block. This is where I am sorry we are not told the Queckenstedt response. Perhaps the low cell count in the cerebro-spinal fluid, no matter what those cells are, supports the concept of a block. But then, even in the uncooperative patient, the pressure of 170 millimetres of cerebro-spinal fluid is not low, and this seems to be against a block.

The chloride is low; but as we have already said, this is of no diagnostic value in this patient. Cerebro-spinal fluid glucose is 48 milligrammes per 100 millilitres and on this I hold rather iconoclastic views. Despite the value which some people have said may accrue from the determination of the cerebro-spinal fluid glucose in tuberculous meningitis, it may be completely normal at any stage of the disease from early to late. If the value had been lower, we might have hung some weight on it. The fact that it is within what I regard as the normal range does not matter a bit.

We are not told the state of the fundi, so we do not know whether she had papilloedema or whether she may have had some miliary tubercules—an outside chance, but still a possibility in a very sick patient.

Her age was quite compatible with the diagnosis; the last three patients with tuberculous meningitis whom I have seen have all been over 50. The blood leucocytosis of 15,000 is quite reasonable for disseminated tuberculosis.

All this suggests that I favour the diagnosis of tuberculous meningitis, which is not so provided the history is accurate. Tuberculous meningitis may be a very acute disease, especially in children. I have known a child die in six days from the apparent onset. In older people such an onset with a very rapid course is unusual. True, the early stages may be insidious and unrecognized, but usually there is something like persistent headache, or loss of powers of concentration or some personality change to point out that all is not well. Here we are rather at a loss in not knowing just how reliable the story of previous good health is.

Again, if this were really an acute case of tuberculous meningitis, we would not expect this cerebro-spinal fluid protein content. In terms of tuberculous meningitis, this protein content indicates chronic changes, and that is incompatible with the history as we are given it.

The second point against the diagnosis of tuberculosis is the close correlation in time between the pulmonary symptoms and the meningeal symptoms. Tuberculous meningitis is due to rupture of an already established caseous focus into the subarachnoid space, and there is no reason why the pulmonary disease should flare up at the same time as the patient develops tuberculous meningitis except as a coincidence. I am not expected to base these conclusions on coincidences, and therefore I must regard the close parallel between the respiratory and nervous system signs as against the diagnosis of tuberculous meningitis. To be sure, an association between miliary and meningeal disease is not uncommon clinically, but on the other hand, miliary tuberculosis does not often lead to hæmoptysis.

I have taken rather a long time to discuss tuberculosis. I think that it had to be excluded as far as we can exclude it, and I think that I was being invited to make that diagnosis.

The second disease in this central nervous system-respiratory group is torulosis, because when we mention tuberculous meningitis it is probably reasonable in many cases to mention torulosis. Again, the main point against this is the very short history. The symptoms and signs, each taken on its own, are quite compatible with torula infection, but the rapid course is very much against it. Pulmonary torulosis in Sydney usually seems to lead to the development of one or two large granulomatous masses in the lungs, and in Peter Harvey's series, if I recall, a diffuse disseminated variety, which might lead to this rapid panting respiration and respiratory distress, constituted less than 10% of cases. Hæmoptysis is a common symptom of torulosis.

Meningeal torulosis may be quite asymptomatic, but even when the onset is fairly abrupt and the patient over a couple of days develops headache and vomiting, the course is rather more prolonged and is usually measured in some weeks if not a month or two. So while I cannot exclude torula for sure, the very acute history is against it.

One other point. If the pathologist does not tell us what the white cells were, then it is very reasonable, without being rude, that he may have made the classical error of mistaking the yeast bodies for white cells.

A third possibility, and not a very likely one, is cerebral abscess *plus* pulmonary suppuration. There is no history of chronic cough or sputum to suggest a diagnosis of bronchiectasis, but this could be an acute lung abscess with a metastatic cerebral focus. The chest story is very rapid even for an acute lung abscess, but the diagnosis is not altogether out of court. Gross hæmoptysis may be a feature of acute lung abscess, even if we do not invoke that very common cause of lung abscess in a person of 67, bronchial carcinoma. If this patient had a cerebral abscess, there are a number of puzzling features. There are no localizing signs at all—that is not incompatible. But to achieve these cerebro-spinal fluid changes and the meningism, we would have to postulate rupture into the subarachnoid space, and I think the cell count of 48 white cells, be they what they may, is rather against this. Again, the very high protein suggests chronicity or loculation, and we have no way of tying this up with a cerebral abscess.

We should mention blood-stream infection with involvement both of lungs and of central nervous system. There is no evidence of endocarditis as a basis. We are told she had no murmurs. This low-grade cerebro-spinal fluid pleocytosis is the sort of thing one sees in a septicæmia, but the protein is very hard to explain on this basis. So I think one can ignore the possibility of a septicæmia or even, with this respiratory and generalized disability, the possibility of "Asian 'flu"—complicated by staphylococcal septicæmia.

The last in this group of infective central nervous system-respiratory diseases is, I think, the most likely one, if we take the first two sentences in the protocol: "The patient was a woman, aged 67 years, whose illness had commenced four days before her admission to hospital, with anoxeria and cough. Soon afterwards she had cold shivers, and the cough became very much worse and productive of blood-tinged sputum." Even to a very young clinician that reads like the story of pneumococcal lobar pneumonia. The sputum, to be sure, was not rusty. It is not always. It may contain frank bright blood, and blood-staining may be considerable. To make the story completely classical it lacks only pleuritic chest pain, and for this we might substitute the upper abdominal tenderness and guarding.

The second part of the history is that of a fulminating meningitis. And this, too, is classically the story of pneumococcal meningitis. All the acute meningitides may have a very acute onset and rapid course; but pneumococcal meningitis is the one *par excellence* which sends people either mad or into coma very quickly. In the usual acute septic meningitis, the cells in the cerebro-spinal fluid are numbered in thousands in the first

12 to 18 hours after the onset. Pneumococcal meningitis is possibly the only exception. In this case they are 48. The cerebro-spinal fluid protein is also compatible with a pneumococcal meningitis of less than 24 hours' duration. I recall one elderly woman who had pneumococcal meningitis and who lived with her relatives, so we knew when the illness started. She had been sick for less than 24 hours, and at lumbar puncture one had to suck the fluid out, it was so thick. It had a protein content of 1950 milligrammes per 100 millilitres, and cells of this order. If this is pneumococcal meningitis, then I would expect these elusive white cells to be all polymorphs.

The last very compatible feature making this likely to be pneumococcal meningitis is the fatal outcome. This, then, is my first diagnosis. Pneumococcal lobar pneumonia, complicated by pneumococcal meningitis.

We must consider a couple of other things briefly to make the story complete. The first possibility is an outside one. Was the patient's central nervous system primarily at fault, and was the patient's condition complicated by a terminal pulmonary infection? I think that we can rule this very quickly out of court, because the course of events is completely against it. We have a clear history of respiratory symptoms and signs coming on before any disturbance of consciousness was sufficiently profound to put the patient in a state where she would be susceptible to a terminal infection. The cerebro-spinal changes are against the diagnosis of such a condition as *tumour cerebri*, and the actual changes, the so-called albumin-cytologic dissociation, would rather suggest a Guillain-Barré type of syndrome. We have absolutely nothing clinically to suggest this. There are no motor changes, and the reflexes were hyperactive rather than hypoactive. I think we must rule out this whole possibility of primary central nervous system disease.

Another possibility just in passing. We mentioned the question earlier, of whether there was significant gastro-intestinal disease. Here we would have to postulate, for example, a carcinoma of the stomach with meningeal seeding, and again a terminal pneumonia. It is not a good choice clinically. The story just does not seem to fit all. It would explain the coffee-grounds vomitus and that is about all. I put it in because it is well known that meningeal seeding with carcinoma, together with tuberculosis and torula, makes up the big group of chronic meningitis. This has the look of a chronic meningitis in terms of the cerebro-spinal fluid changes if not clinically. But the acuteness of the whole story would strain the credulity of a diagnosis of gastric carcinoma.

Is there significant renal disease? We know she had albuminuria. We do not know how much. If it was only a little, it may have been toxic or febrile. If there was a lot, it may favour renal disease. The specific gravity of the urine was 1·020, which shows (in the absence of glucose in that urine to weigh it) that she can concentrate to that degree in response to dehydration. I am pinning my faith to the specific gravity and, assuming that it is accurate, I do not think this patient had significant renal disease. If she did, we would have to postulate, say, chronic pyelonephritis, with terminal uræmia and a liability to infection. The uræmia *per se* may lead to leucocytosis and to the vomiting of blood, but I do not think I have enough evidence to incriminate the kidneys at all, and I do not intend to complicate the issue.

Had she *diabetes mellitus*? Again we do not know, because we have not sufficient evidence. Like uræmia, I do not think it is likely to be primary. She may well have had *diabetes mellitus* with a high renal threshold, or even with Kimmelstiel-Wilson disease. On the other hand, we can advance other very good causes for the ketonuria—starvation and dehydration. We could even advance other causes for the glycosuria later in the course of the disease. It may have been secondary to intracranial disturbances. She was being resuscitated, so no doubt she was being given a lot of glucose intravenously. So we can suggest other reasons than diabetes why she may have had sugar in the urine. I am ruling out renal disease and I am ruling out *diabetes mellitus*, both on the basis of absence of evidence.

That is about all I have to say. If I may just ride one hobby-horse, because we must put an outsider in the field. It is the only collagen disease which I am going to introduce—so-called pathergic or Wegener's granulomatosis which, as you know, is in some ways related to *polyarteritis nodosa*. Respiratory involvement is universal. They frequently have hæmoptysis. They frequently have high fever with chills. Renal involvement is manifest by albuminuria, hæmaturia and progressive azotæmia. The course is usually acute or subacute, but not usually as acute as this. Secondary infection and bleeding phenomena from the bowel and from other sites are not uncommon. I have not seen a case with central nervous system involvement, but they are described, either in the form of discrete granulomata, or with a widespread vasculitis. At least with a widespread vasculitis you might get these cerebro-spinal fluid changes. Usually, however, they have focal signs due to granulomata. This woman had none. I think I have said enough about Wegener's granulomatosis to indicate that the story is not altogether incompatible with the story in this case, and I am certainly not going to push the diagnosis.

So Mr. Chairman, my first diagnosis would be, if the story is accurate, pneumococcal pneumonia with pneumococcal meningitis. But if we may distrust the history a little, I would like to put second and third tuberculous meningitis and torulosis; and I would like to put in Wegener's granulomatosis as the outsider.

DR. EVANS: I would like to thank Dr. Read for a masterly discussion of a very difficult patient, and now the subject is open for discussion. I think I would call on the physicians first to give us their view. There are bound to be divergent views on a protocol of this type, so that I wonder if any of the physicians would care to join in—or some of the resident staff.

DR. W. H. WOLFENDEN: I do not think I have anything to add to that very masterly discussion of the problem, sir. I think I agree with the idea that this condition has been basically one of pulmonary infection, with secondary involvement of the nervous system. A cerebro-spinal fluid protein of this type is one that might occur in a cerebro-spinal fluid block, and is what one would expect in primary disease of the central nervous system. The history, as has been suggested, is one of the acute onset of respiratory illness, and I agree wholeheartedly with that. Primary respiratory illness and nervous system involvement.

DR. J. LANCE: I would like to comment, sir, on the very high cerebro-spinal fluid protein, which is a very interesting point on the neurological side. I was not aware that in pneumococcal meningitis you could get these very high figures with such a low cell count, and I was wondering if possibly there could be something else hidden away in this story which gave an incidental finding of a very high protein, or if there was a hidden eighth nerve tumour, or something of that sort, the sort of red herring that one gets at these clinico-pathological conferences.

Spinal block is, of course, the great cause of such a high protein, and in the Guillain-Barré syndrome it is œdema of the nerve roots around the affected roots which gives the block of absorption and increases the protein in the sac. I was wondering, too, if there could be some other cause of block in this patient. It could possibly be part of a chronic tuberculous meningitis if only the story was a little longer. So I think that is a very interesting thing that remains to be explained.

The other thing is the low chloride in the cerebro-spinal fluid, which the speaker said could possibly be discounted. This patient is dehydrated as far as we can see, so it is quite likely that this finding is significant in this patient. I was wondering if it was possible that she had a septicæmic process that involved the adrenals, some sort of adrenal or Waterhouse-Friderichsen type of collapse, and an Addisonian picture producing the low chloride—that is if we do not consider tuberculous meningitis as being a cause of low chlorides, or pneumonia, which I understand is also a cause of it.

DR. EVANS: Would Dr. Read care to comment on that?

DR. READ: I would only like to say that it would have been nice to know the blood pressure, as far as one of his suggestions was concerned.

DR. E. HIRST: I am sorry about that, it was an omission. The blood pressure was 160/90 millimetres of mercury. We do not have any details of the other things that you ask for, nor an X ray of the chest.

DR. READ: I think Dr. Lance's point about her being dehydrated, and questioning my explanation of the low chloride, is a very reasonable one. I do not quite know where to go from there.

DR. EVANS: Would any other members of the resident medical staff care to speak? Dr. Handley perhaps.

DR. D. HANDLEY: I do not think that I have anything very much to add to that. I think that the high protein and low cells in the cerebro-spinal fluid is compatible with a pneumococcal meningitis. I have seen one case in which there were not

malaise. She may well have had chronic pulmonary tuberculosis with or without a recent acute spread. The coffee-grounds vomitus might well come from swallowed blood. We have seen patients with large cavities in their lungs due to tuberculosis, who are coughing up a little blood, vomiting quite a lot of altered blood, and at autopsy there is no lesion in the stomach to account for the bleeding. We can only assume that they are swallowing more blood than they are coughing up. So all this is compatible with pulmonary tuberculosis. What about tuberculous meningitis? We know that she had meningitis with a low cell count in the cerebro-spinal fluid. The cells in tuberculous meningitis may number anything from a few to over a thousand. The type does not matter a bit. They may be all polymorphs or all lymphocytes or any mixture of the two at any stage of the disease. More commonly they are predominantly polymorphs during the so-called intrathecal tuberculin reaction after treatment has commenced. But even in the untreated disease there may be spontaneous bursts of almost pure polymorphs. So at this point I do not particularly mind what the leucocytes were.

The rise of cerebro-spinal fluid protein is again classical of tuberculous meningitis, but in acute cases one would like it to be rather closer to 100 than to 1000 milligrammes per 100 millilitres. In the straightforward case of tuberculous meningitis, the level is usually around several hundred milligrammes per 100 millilitres. The level, of course, may be very high. I have seen one case where the cerebro-spinal fluid protein level reached 3·5 grammes per 100 millilitres, and a record of another case in which it reached 11 grammes per 100 millilitres. Such levels as these, or even one of 1000 milligrammes per 100 millilitres, are usually associated with loculation in the subarachnoid space and the presence of subarachnoid block. Indeed, in recent years some of us have regarded a high or a rising cerebro-spinal fluid protein in tuberculous meningitis as an indication for steroid therapy. So if this is tuberculous meningitis, a cerebro-spinal fluid protein of 1000 milligrammes per 100 millilitres suggests a block. This is where I am sorry we are not told the Queckenstedt response. Perhaps the low cell count in the cerebro-spinal fluid, no matter what those cells are, supports the concept of a block. But then, even in the uncooperative patient, the pressure of 170 millimetres of cerebro-spinal fluid is not low, and this seems to be against a block.

The chloride is low; but as we have already said, this is of no diagnostic value in this patient. Cerebro-spinal fluid glucose is 48 milligrammes per 100 millilitres and on this I hold rather iconoclastic views. Despite the value which some people have said may accrue from the determination of the cerebro-spinal fluid glucose in tuberculous meningitis, it may be completely normal at any stage of the disease from early to late. If the value had been lower, we might have hung some weight on it. The fact that it is within what I regard as the normal range does not matter a bit.

We are not told the state of the fundi, so we do not know whether she had papilloedema or whether she may have had some miliary tubercules—an outside chance, but still a possibility in a very sick patient.

Her age was quite compatible with the diagnosis; the last three patients with tuberculous meningitis whom I have seen have all been over 50. The blood leucocytosis of 15,000 is quite reasonable for disseminated tuberculosis.

All this suggests that I favour the diagnosis of tuberculous meningitis, which is not so provided the history is accurate. Tuberculous meningitis may be a very acute disease, especially in children. I have known a child die in six days from the apparent onset. In older people such an onset with a very rapid course is unusual. True, the early stages may be insidious and unrecognized, but usually there is something like persistent headache, or loss of powers of concentration or some personality change to point out that all is not well. Here we are rather at a loss in not knowing just how reliable the story of previous good health is.

Again, if this were really an acute case of tuberculous meningitis, we would not expect this cerebro-spinal fluid protein content. In terms of tuberculous meningitis, this protein content indicates chronic changes, and that is incompatible with the history as we are given it.

The second point against the diagnosis of tuberculosis is the close correlation in time between the pulmonary symptoms and the meningeal symptoms. Tuberculous meningitis is due to rupture of an already established caseous focus into the subarachnoid space, and there is no reason why the pulmonary disease should flare up at the same time as the patient develops tuberculous meningitis except as a coincidence. I am not expected to base these conclusions on coincidences, and therefore I must regard the close parallel between the respiratory and nervous system signs as against the diagnosis of tuberculous meningitis. To be sure, an association between miliary and meningeal disease is not uncommon clinically, but on the other hand, miliary tuberculosis does not often lead to hæmoptysis.

I have taken rather a long time to discuss tuberculosis. I think that it had to be excluded as far as we can exclude it, and I think that I was being invited to make that diagnosis.

The second disease in this central nervous system-respiratory group is torulosis, because when we mention tuberculous meningitis it is probably reasonable in many cases to mention torulosis. Again, the main point against this is the very short history. The symptoms and signs, each taken on its own, are quite compatible with torula infection, but the rapid course is very much against it. Pulmonary torulosis in Sydney usually seems to lead to the development of one or two large granulomatous masses in the lungs, and in Peter Harvey's series, if I recall, a diffuse disseminated variety, which might lead to this rapid panting respiration and respiratory distress, constituted less than 10% of cases. Hæmoptysis is a common symptom of torulosis.

Meningeal torulosis may be quite asymptomatic, but even when the onset is fairly abrupt and the patient over a couple of days develops headache and vomiting, the course is rather more prolonged and is usually measured in some weeks if not a month or two. So while I cannot exclude torula for sure, the very acute history is against it.

One other point. If the pathologist does not tell us what the white cells were, then it is very reasonable, without being rude, that he may have made the classical error of mistaking the yeast bodies for white cells.

A third possibility, and not a very likely one, is cerebral abscess *plus* pulmonary suppuration. There is no history of chronic cough or sputum to suggest a diagnosis of bronchiectasis, but this could be an acute lung abscess with a metastatic cerebral focus. The chest story is very rapid even for an acute lung abscess, but the diagnosis is not altogether out of court. Gross hæmoptysis may be a feature of acute lung abscess, even if we do not invoke that very common cause of lung abscess in a person of 67, bronchial carcinoma. If this patient had a cerebral abscess, there are a number of puzzling features. There are no localizing signs at all—that is not incompatible. But to achieve these cerebro-spinal fluid changes and the meningism, we would have to postulate rupture into the subarachnoid space, and I think the cell count of 48 white cells, be they what they may, is rather against this. Again, the very high protein suggests chronicity or loculation, and we have no way of tying this up with a cerebral abscess.

We should mention blood-stream infection with involvement both of lungs and of central nervous system. There is no evidence of endocarditis as a basis. We are told she had no murmurs. This low-grade cerebro-spinal fluid pleocytosis is the sort of thing one sees in a septicæmia, but the protein is very hard to explain on this basis. So I think one can ignore the possibility of a septicæmia or even, with this respiratory and generalized disability, the possibility of "Asian 'flu"—complicated by staphylococcal septicæmia.

The last in this group of infective central nervous system-respiratory diseases is, I think, the most likely one, if we take the first two sentences in the protocol: "The patient was a woman, aged 67 years, whose illness had commenced four days before her admission to hospital, with anoxeria and cough. Soon afterwards she had cold shivers, and the cough became very much worse and productive of blood-tinged sputum." Even to a very young clinician that reads like the story of pneumococcal lobar pneumonia. The sputum, to be sure, was not rusty. It is not always. It may contain frank bright blood, and blood-staining may be considerable. To make the story completely classical it lacks only pleuritic chest pain, and for this we might substitute the upper abdominal tenderness and guarding.

The second part of the history is that of a fulminating meningitis. And this, too, is classically the story of pneumococcal meningitis. All the acute meningitides may have a very acute onset and rapid course; but pneumococcal meningitis is the one *par excellence* which sends people either mad or into coma very quickly. In the usual acute septic meningitis, the cells in the cerebro-spinal fluid are numbered in thousands in the first

12 to 18 hours after the onset. Pneumococcal meningitis is possibly the only exception. In this case they are 48. The cerebro-spinal fluid protein is also compatible with a pneumococcal meningitis of less than 24 hours' duration. I recall one elderly woman who had pneumococcal meningitis and who lived with her relatives, so we knew when the illness started. She had been sick for less than 24 hours, and at lumbar puncture one had to suck the fluid out, it was so thick. It had a protein content of 1950 milligrammes per 100 millilitres, and cells of this order. If this is pneumococcal meningitis, then I would expect these elusive white cells to be all polymorphs.

The last very compatible feature making this likely to be pneumococcal meningitis is the fatal outcome. This, then, is my first diagnosis. Pneumococcal lobar pneumonia, complicated by pneumococcal meningitis.

We must consider a couple of other things briefly to make the story complete. The first possibility is an outside one. Was the patient's central nervous system primarily at fault, and was the patient's condition complicated by a terminal pulmonary infection? I think that we can rule this very quickly out of court, because the course of events is completely against it. We have a clear history of respiratory symptoms and signs coming on before any disturbance of consciousness was sufficiently profound to put the patient in a state where she would be susceptible to a terminal infection. The cerebro-spinal changes are against the diagnosis of such a condition as *tumour cerebri*, and the actual changes, the so-called albumin-cytologic dissociation, would rather suggest a Guillain-Barré type of syndrome. We have absolutely nothing clinically to suggest this. There are no motor changes, and the reflexes were hyperactive rather than hypoactive. I think we must rule out this whole possibility of primary central nervous system disease.

Another possibility just in passing. We mentioned the question earlier, of whether there was significant gastro-intestinal disease. Here we would have to postulate, for example, a carcinoma of the stomach with meningeal seeding, and again a terminal pneumonia. It is not a good choice clinically. The story just does not seem to fit all. It would explain the coffee-grounds vomitus and that is about all. I put it in because it is well known that meningeal seeding with carcinoma, together with tuberculosis and torula, makes up the big group of chronic meningitis. This has the look of a chronic meningitis in terms of the cerebro-spinal fluid changes if not clinically. But the acuteness of the whole story would strain the credulity of a diagnosis of gastric carcinoma.

Is there significant renal disease? We know she had albuminuria. We do not know how much. If it was only a little, it may have been toxic or febrile. If there was a lot, it may favour renal disease. The specific gravity of the urine was 1·020, which shows (in the absence of glucose in that urine to weigh it) that she can concentrate to that degree in response to dehydration. I am pinning my faith to the specific gravity and, assuming that it is accurate, I do not think this patient had significant renal disease. If she did, we would have to postulate, say, chronic pyelonephritis, with terminal uræmia and a liability to infection. The uræmia *per se* may lead to leucocytosis and to the vomiting of blood, but I do not think I have enough evidence to incriminate the kidneys at all, and I do not intend to complicate the issue.

Had she *diabetes mellitus*? Again we do not know, because we have not sufficient evidence. Like uræmia, I do not think it is likely to be primary. She may well have had *diabetes mellitus* with a high renal threshold, or even with Kimmelstiel-Wilson disease. On the other hand, we can advance other very good causes for the ketonuria—starvation and dehydration. We could even advance other causes for the glycosuria later in the course of the disease. It may have been secondary to intracranial disturbances. She was being resuscitated, so no doubt she was being given a lot of glucose intravenously. So we can suggest other reasons than diabetes why she may have had sugar in the urine. I am ruling out renal disease and I am ruling out *diabetes mellitus*, both on the basis of absence of evidence.

That is about all I have to say. If I may just ride one hobby-horse, because we must put an outsider in the field. It is the only collagen disease which I am going to introduce—so-called pathergic or Wegener's granulomatosis which, as you know, is in some ways related to *polyarteritis nodosa*. Respiratory involvement is universal. They frequently have hæmoptysis. They frequently have high fever with chills. Renal involvement is manifest by albuminuria, hæmaturia and progressive azotæmia. The course is usually acute or subacute, but not usually as acute as this. Secondary infection and bleeding phenomena from the bowel and from other sites are not uncommon. I have not seen a case with central nervous system involvement, but they are described, either in the form of discrete granulomata, or with a widespread vasculitis. At least with a widespread vasculitis you might get these cerebro-spinal fluid changes. Usually, however, they have focal signs due to granulomata. This woman had none. I think I have said enough about Wegener's granulomatosis to indicate that the story is not altogether incompatible with the story in this case, and I am certainly not going to push the diagnosis.

So Mr. Chairman, my first diagnosis would be, if the story is accurate, pneumococcal pneumonia with pneumococcal meningitis. But if we may distrust the history a little, I would like to put second and third tuberculous meningitis and torulosis; and I would like to put in Wegener's granulomatosis as the outsider.

DR. EVANS: I would like to thank Dr. Read for a masterly discussion of a very difficult patient, and now the subject is open for discussion. I think I would call on the physicians first to give us their view. There are bound to be divergent views on a protocol of this type, so that I wonder if any of the physicians would care to join in—or some of the resident staff.

DR. W. H. WOLFENDEN: I do not think I have anything to add to that very masterly discussion of the problem, sir. I think I agree with the idea that this condition has been basically one of pulmonary infection, with secondary involvement of the nervous system. A cerebro-spinal fluid protein of this type is one that might occur in a cerebro-spinal fluid block, and is what one would expect in primary disease of the central nervous system. The history, as has been suggested, is one of the acute onset of respiratory illness, and I agree wholeheartedly with that. Primary respiratory illness and nervous system involvement.

DR. J. LANCE: I would like to comment, sir, on the very high cerebro-spinal fluid protein, which is a very interesting point on the neurological side. I was not aware that in pneumococcal meningitis you could get these very high figures with such a low cell count, and I was wondering if possibly there could be something else hidden away in this story which gave an incidental finding of a very high protein, or if there was a hidden eighth nerve tumour, or something of that sort, the sort of red herring that one gets at these clinico-pathological conferences.

Spinal block is, of course, the great cause of such a high protein, and in the Guillain-Barré syndrome it is œdema of the nerve roots around the affected roots which gives the block of absorption and increases the protein in the sac. I was wondering, too, if there could be some other cause of block in this patient. It could possibly be part of a chronic tuberculous meningitis if only the story was a little longer. So I think that is a very interesting thing that remains to be explained.

The other thing is the low chloride in the cerebro-spinal fluid, which the speaker said could possibly be discounted. This patient is dehydrated as far as we can see, so it is quite likely that this finding is significant in this patient. I was wondering if it was possible that she had a septicæmic process that involved the adrenals, some sort of adrenal or Waterhouse-Friderichsen type of collapse, and an Addisonian picture producing the low chloride—that is if we do not consider tuberculous meningitis as being a cause of low chlorides, or pneumonia, which I understand is also a cause of it.

DR. EVANS: Would Dr. Read care to comment on that?

DR. READ: I would only like to say that it would have been nice to know the blood pressure, as far as one of his suggestions was concerned.

DR. E. HIRST: I am sorry about that, it was an omission. The blood pressure was 160/90 millimetres of mercury. We do not have any details of the other things that you ask for, nor an X ray of the chest.

DR. READ: I think Dr. Lance's point about her being dehydrated, and questioning my explanation of the low chloride, is a very reasonable one. I do not quite know where to go from there.

DR. EVANS: Would any other members of the resident medical staff care to speak? Dr. Handley perhaps.

DR. D. HANDLEY: I do not think that I have anything very much to add to that. I think that the high protein and low cells in the cerebro-spinal fluid is compatible with a pneumococcal meningitis. I have seen one case in which there were not

any cells at all. The protein was quite high and in that case the smear was crowded with the pneumococcal bacteria. On the protocol I think that that might well have been the diagnosis in the first place—pneumococcal meningitis in the fulminating form where there are not many cells, with a short history, and where there are often a lot of bacteria in the culture in the cerebro-spinal fluid. We have not got a chest X-ray certainly, but what information we do have does suggest that the infection was primarily in the chest.

I think that another possibility is a staphylococcal septicæmia, a staphylococcal pneumonia with spread to the subarachnoid space, although as Dr. Read said they often do have an acute endocarditis at the same time. I think the story is a bit short for a tuberculous meningitis unless there is some other story to it.

DR. EVANS: I see Dr. Hurt sitting there as if he disagrees with some of the things that have been said. Dr. Hurt?

DR. B. M. HURT: I disagree, sir, with Dr. Read's dismissal of lung signs as a secondary consideration, in so far as often one does not have a portable X-ray machine to carry around in one's bag. I think that, no matter how we look at it, we still must regard the four signs of clinical diagnosis as primarily useful in chest diagnosis. Things such as fluid in the chest can be readily detected by clinical signs. I would not have been quite as iconoclastic on that point, especially if there are any final-year students here this afternoon who will be expected in a few weeks' time to demonstrate these things. I do not disagree that notable abnormalities can be present in the lungs and cannot be detected by clinical signs.

The second point: Dr. Handley stole my thunder a little by discussing the pneumococcal culture in the cerebro-spinal fluid, because I have, to my satisfaction, demonstrated to a non-believer bile solubility in a primary cerebro-spinal fluid take-off without having to go through culture. But it is true that if you have a cloudy cerebro-spinal fluid and a few white cells, then you can often see the lanceolate diplococci and demonstrate bile solubility. I think in practice that this lady did have pneumococcal meningitis and a primary pneumococcal pneumonia. If we take a small point and say that the staff was correct in their diagnosis, they directed their treatment to a bacterial non-tuberculous meningitis, by giving penicillin intrathecally, "Trisulpha" and tetracycline.

I thought originally, when I read this protocol, that we were dealing here with an abscess in the brain because of the glucose in the cerebro-spinal fluid. However, if this lady was a diabetic and had an originally very high glucose, this may in fact represent a reduction in her cerebro-spinal fluid glucose; but Dr. Read has eased this in my mind by saying that if this was fairly rapid, the glucose may have not all been absorbed by the bacteria themselves.

So I commend Dr. Read on his summary and agree that this woman had pneumococcal meningitis.

DR. EVANS: I wonder if our senior physician, Dr. Noad, would care to give us his views.

DR. K. B. NOAD: I would like to commend your idea of hearing from the junior physicians. I think that there is very little to be added to their statements. In regard to these pure cultures of pneumococci in the cerebro-spinal fluid, the idea does not seem to be compatible with a glucose of 48, because we have always imagined that the organisms batten on the glucose in the fluid; but, however, I have nothing further to add to this discussion.

DR. EVANS: If there are no other comments, we had better ask Dr. Hirst to give us the story.

Autopsy Report.

DR. E. HIRST: The patient was a thin woman of about the stated age, and the relevant findings were as follows:

The right pleural cavity was completely obliterated by dense fibrous adhesions. The left pleural cavity was normal. There was frothy blood-stained sputum in the trachea. The right middle lobe was consolidated. It was uniformly grey in colour, firm and friable, and portion of it sank in the fixative. The right upper and lower lobes and left lung showed varying degrees of congestion.

The brain showed a pale-green, purulent exudate in the subarachnoid space, prominent at the base and extending over the hemispheres especially along the lines of the middle cerebral vessels. The ventricles contained turbid cerebro-spinal fluid.

The mucosa of the stomach was congested and dark and contained coffee-grounds material. The contents of the small intestine were also blood-stained. In the autopsy protocol there is no mention made of erosions of the gastric mucosa, but I take it that the state of the gastric contents was due to hæmorrhagic erosions in the gastric mucosa.

The kidneys showed only post-mortem changes. There was difficulty in deciding whether there was any evidence of tubular damage. We thought not. There was no evidence of *diabetes mellitus* in the post-mortem material. Microscopic examination of sections of the lung and meninges confirmed the diagnosis of lobar pneumonia and suppurative meningitis.

Moreover, from the cerebro-spinal fluid during life, a heavy culture of *Streptococcus pneumoniæ* was obtained.

The final diagnosis was pneumococcal pneumonia and suppurative meningitis.

Pathological Discussion.

DR. EVANS: It only remains that we congratulate Dr. Read on his excellent diagnosis. The physicians have really fulfilled their purpose in this particular case. Would anyone like to comment any further on the findings at *post mortem*?

DR. HANDLEY: It seems strange to me that there is so much pus present in the meninges and yet so few cells in the cerebro-spinal fluid itself. The meninges were considerably swollen and contained a lot of pus. I would have thought that there would be more pus in the cerebro-spinal fluid unless it was loculated.

DR. HIRST: All I can say is that this cerebro-spinal fluid was examined by a fairly experienced person, and I think that those findings represent the true findings in this cerebro-spinal fluid.

PROFESSOR F. R. MAGAREY: I think those findings are quite compatible with loculation, with a very thick purulent exudate. The cells are prevented by the exudate from getting down to the lumbar region, but the protein can get through.

DR. J. BLACKWELL: Was a positive culture obtained during life?

DR. HIRST: Yes, this culture was of the cerebro-spinal fluid during life.

DR. HURT: What is a good method of demonstrating a differential cell count on a cerebro-spinal fluid?

DR. BLACKWELL: I can recommend to Dr. Hurt the method that is used in our laboratory, that is staining with 0·2% aqueous solution of toluidine blue. One drop of toluidine blue is added to nine drops of cerebro-spinal fluid in a centrifuge tube. This is shaken and allowed to stand for approximately five minutes. We then run the fluid on to a counting chamber, on which we can then do a differential and total count. We find it is fairly easy to distinguish the polymorphs, mononuclears and red cells.

Diagnosis.

Pneumococcal lobar pneumonia and suppurative meningitis.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

SALARIES AT SYDNEY HOSPITAL.

[From the *Australasian Medical Gazette*, January, 1893.]

THE Directors of the Sydney Hospital have agreed to the following recommendations of the subcommittee appointed to inquire into the financial working of the institution.

1. That the salary of the Medical Superintendent should be £300 *per annum* with board and quarters instead of £500, the present salary. They are of opinion that the tenure of this office should be fixed at three years and that the officer be eligible but not entitled to re-election.

2. That only one of the Resident Medical Officers should receive the present rate of salary, viz., £100 *per annum*. This officer should be called the Senior Resident Medical Officer and his duties should be to give the anæsthetics

as far as possible and to take the place of the Medical Superintendent in his absence. The salary of the other Resident Medical Officers should be reduced to £50 *per annum* and the Committee recommend that the Resident Medical Officers only hold office for one year. As regards the out-patient department, the Committee recommend that the Regent Street Dispensary be closed, that the present system of paying District Surgeons should be abolished, and that the work should be done by the Assistant Honorary Medical Officers.

Special Correspondence.

LONDON LETTER.

BY OUR SPECIAL CORRESPONDENT.

London Teaching Hospitals.

PLANS for the rebuilding of St. Thomas's Hospital, grievously damaged in the war, have begun—a project which will drastically transform the area of the South Bank of the Thames opposite the Houses of Parliament. A new hospital of 827 beds will rise on the site in marked contrast in design from the old style of ward pavilions abutting the river. The total building cost is roughly estimated at seven million pounds, and the operation will take from nine to twelve years. The area of the new hospital will be approximately double the present one, obtained by acquiring additional land and by a diversion of Lambeth Palace Road. Construction is planned to take place in four stages so that the work of the hospital will not be upset. Provision is also made for a new medical school. The total cost will be a government responsibility, and the Minister of Health has given his agreement to the scheme.

Many teaching hospitals in London occupy valuable central sites where expansion is difficult and conditions are far from ideal for patients. Charing Cross Hospital, just off the Strand, and St. George's Hospital, at Hyde Park Corner, have for some years been considering moving out of central London. Charing Cross Hospital planned to rebuild at Harrow in Middlesex, a growing area in need of a first-class hospital, but the scheme was vetoed by London University on the grounds that Harrow was too far from the University for the students. Last year it was proposed to rebuild Charing Cross Hospital at Fulham, incorporating the local hospital, but strong local opposition has been encountered, and no decision has yet been reached. St. George's Hospital has plans for rebuilding at Tooting where 30 acres are available. A proposal that St. George's and Charing Cross Hospitals should amalgamate was not found acceptable, as neither wanted to lose its identity. Equally unacceptable was the suggestion that both should amalgamate with Westminster Hospital to form one large teaching hospital.

Outside the central London area, the peripheral hospitals have so improved in facilities, with first-class consultant staffs, that patients are increasingly referred to them by general practitioners. The result is that the London teaching hospitals are rapidly losing the material they need for teaching, and are appealing to the family doctors to send them cases.

Mental Illness.

The attitude of the general public towards mental illness is gradually changing. Despite the advances in the treatment of mental disease in recent years, workers in this field have had to struggle against ignorance and superstition, which still lingers among some sections of the public. The enlightened policy of education pursued by central and local health departments, aided by the Press and the B.B.C. through the medium of television, has led to greater understanding and sympathy for the mentally afflicted. The aim is to convince the public that they should look on a mental hospital as they do upon an ordinary hospital for the physically sick. Visible evidence of the success of the new approach is seen in the number of voluntary as opposed to certified patients admitted to mental institutions. In 1956, 78·2% of admissions to mental hospitals were voluntary, while the total number of certified cases fell. Mental illness and mental institutions have been for too long isolated from the rest of the community.

Mental hospitals are still overcrowded and handicapped by out-of-date buildings not easily adaptable to modern medical and surgical needs, and by chronic staff shortages. Much publicity has been given to the conditions in some institutions, which has led to calls for more and improved accommodation for mental patients. The Minister of Health has given priority to building projects for enlarging and adapting existing accommodation, and the first new hospital to be built in this country since the commencement of the National Health Service is a mental institution. Some easement in the situation has occurred, as the total number of admissions to mental hospitals has been falling over the past few years.

The view that admission to hospital is not necessary for many psychiatric patients has been put forward by Dr. J. Carse and his colleagues of Graylingwell Mental Hospital, Chichester, as the result of a two-year experiment on the out-patient and domiciliary treatment of the mental patients in the catchment area of the hospital. During the first 10 months, when the scheme was operating, the number of patients admitted to the mental hospitals decreased by 59% compared with the same period in 1956, whereas the number of patients admitted from other areas rose by 4%. The conclusion is reached that overcrowding in mental hospitals could be entirely removed by the provision of an out-patient and domiciliary service, although there will always be a need for accommodation for patients who need constant care and attention and for those whose treatment cannot be carried out in clinics or in the home. Commenting on this experiment, *The Lancet* was sufficiently optimistic to state: "Within 10 years, we will be wondering what to do with the many mental hospitals, which are plainly unsuitable for the proper practice of psychiatry within the same frame-work as the rest of medicine."

The new developments in the treatment of mental disease will require changes in the existing law relating to mental illness and mental deficiency. The Government are now studying the report of the Royal Commission on this aspect and have accepted the need for a revision of the law, but many questions of principle and detail have still to be worked out before legislation can be introduced to Parliament. One recommendation of the Royal Commission not requiring legislative action has already been acted upon in regard to mental defectives—namely, admission to hospital without certification and the removal of detention in selected cases.

The success of training establishments for the mentally handicapped has undoubtedly influenced this decision. The number of centres for the training of mental defectives in Great Britain has increased threefold since the beginning of the National Health Service. Today, over nine thousand children under 16 years of age are receiving training, although there are still over three thousand suitable for training but not receiving it, owing to the national financial stringency. The more complicated problem of adult defectives, of whom there are about five thousand, has not yet been properly studied, although a few adult training centres have been established.

Opening a new occupational centre at Chelmsford last month, the Minister of Health stated: "There is no justification, medical, moral, or social, for the life-long separation of the mentally handicapped from society, when training can make many of them acceptable members of it."

Occupational Mortality.

Many different factors operate in the selection of an occupation by an individual—i.e., physical fitness, parental influence, geographical position—and these factors must all to a greater or less extent affect mortality. The extent to which these factors operate in different occupations must be borne in mind before it may be inferred that the increased death rate is necessarily due to the occupation itself. These remarks accompany a recent report of the General Register Office on an analysis of mortality by occupations, which is based on the deaths in the years 1948 to 1953, and provides the most recent information for those concerned with occupation and environment in relation to health. The greater part of the report is concerned with the deaths of men aged 20 to 64 years, since it is in this range that any deleterious effect of a man's occupation is most likely to be seen. The effect of occupation on a man's health cannot be separated from other environmental factors, e.g., his living conditions. Married women, on the other hand, share their husbands' environment in most respects except occupation. By analysing the mortality of married women, grouped according to their husband's occupation, it is justifiable to assume that, if their mortality is less than the husbands', the excess of mortality among the men may be associated with the occupation itself.

All occupations have been assigned in the report to five broad social groups: (i) professional occupations, e.g., law, medicine, the Church; (ii) managerial occupations, e.g., employers, managers, farmers; (iii) skilled occupations, e.g., fitters, engine drivers, clerks; (iv) semi-skilled occupations,

e.g., machine minders; (v) unskilled occupations, e.g., labourers, kitchen hands.

Compared with former censuses, those in group (ii) (the employers and managers) now occupy the most favourable position, and those in group (iv) come next.

Some interesting points emerge from the study of mortality in individual occupations.

Doctors have a rather similar mortality to clergymen, but their death rate from coronary and cerebro-vascular disease is higher. They have a relatively high death rate from suicide. Although the number of deaths was small, there is a high risk of death from poliomyelitis. This might have been due to excessive contact with infectious patients, but it must be compared with the almost equally high rate from the same cause among the legal profession.

Clergymen of all denominations, except Roman Catholic priests, have favourable death rates, although they share a high mortality with others engaged in professional occupations, from coronary and cerebro-vascular disease.

Teachers have an even lower mortality than clergymen or doctors; it is particularly low from tuberculosis.

Senior civil servants have a rather high death rate from coronary disease, which would appear to be earlier in onset than in their counterparts in commerce.

Farmers and farm labourers generally have a low mortality.

Coal miners have a high death rate from accidents, and the risk of industrial lung disease is well known. Mortality from cancer of the lung is very low and also that from poliomyelitis.

Foundry men and furnace men have a comparatively high death rate from respiratory diseases and cancer of the lung, which is counterbalanced by a low death rate from heart disease.

Boot and shoe makers are shown to have a high death rate from tuberculosis, in common with dock labourers.

Printers and bookbinders have an apparently high death rate from leuksemia, for which it is difficult to suggest a cause.

Publicans and barmen are groups which have some of the highest death rates, particularly from cancer of the respiratory and alimentary tracts, from vascular lesions of the central nervous system and from cirrhosis of the liver.

An analysis of some of the important causes of death according to the social group have also been made. Tuberculosis, syphilis, cancer of the stomach, bronchitis and pneumonia are all diseases which have a steady rise in mortality from a low death rate in group (i) to a high rate in group (v). For men only, the same is true of cancer of the lung.

Diseases which show the opposite tendency, with a high mortality in group (i) and a downward trend to group (v), are poliomyelitis in both sexes, coronary disease in males and cancer of the breast in women. The point is made that a high standard of living carries its own particular mortality risk.

The Control of Cancer.

During the last twenty years, the total mortality from cancer has increased at every age, but has declined among middle-aged women. The difference between the sexes is due to cancer of the lung, which, while increasing in both sexes, affects more men than women. If we exclude cancer of the lung, death rates from cancer have declined in both sexes, except among children and very old persons.

For the period 1950-1954, the forms of cancer by site causing most deaths in males were as follows (the rate per million of population is shown in parentheses): lung (336), stomach (211), intestine (109), rectum (88), prostate (74). The sites and rates in females were: breast (182), stomach (118), intestine (106), uterus (91), ovary (60).

Deaths of children under 15 years of age from cancer (over half due to malignant disease of blood-forming organs) represent 1% of the total cancer deaths.

In 1956, a total of 92,710 persons died of cancer in England and Wales (48,935 males and 43,775 females); 20% of these died from cancer of the lung.

Recent work on the relationship of cigarette smoking to lung cancer has served to confirm earlier views that there is an association between the two, although the precise nature has not yet been determined. There appear to be, in Great Britain, other factors besides the smoking habit—factors associated with urbanization, with its accompanying pollution of the atmosphere by smoke and the waste products of the internal combustion engine. This work represents a new approach to the problem on the causation of cancer of the lung, which has been successfully applied in the past to the prevention of certain forms of occupational cancer.

The fundamental cause of cancer still remains. While basic medical research endeavours to unlock the secrets of the living cell, the epidemiological approach is receiving increasing attention, with the object of ascertaining the factors, often preventible, which initiate or perpetuate the cancerous process.

Obituary.

JOHN O'SULLIVAN.

WE are indebted to Dr. Kevin O'Day for the following account of the career of the late Dr. John O'Sullivan.

The sudden death of John O'Sullivan has robbed radiology of one of its most able and most picturesque personalities. From his earliest student days John O'Sullivan displayed a singleness and tenacity of purpose which were eventually to place him in the front rank of his specialty. It is no exaggeration to say that, in a difficult case, finality had not been reached until "Johnny" had seen the films, and beyond him there was no further court of appeal. His almost reckless expenditure of film was a symbol of his tireless pursuit of truth, and this was not limited to radiology.

John O'Sullivan received his early education at the Christian Brothers' College, North Melbourne, and graduated from the University of Melbourne in 1921. He was a student, resident and registrar at St. Vincent's Hospital, Melbourne, and later became honorary diagnostic and therapeutic radiologist. Under his able direction the X-ray Department at St. Vincent's kept pace with modern development and contributed in no small measure to the rapid growth of the clinical school of the hospital and to the position it now holds in the field of medicine in Melbourne. In the year preceding his retirement he rounded off his service to the hospital by becoming chairman of the honorary staff.

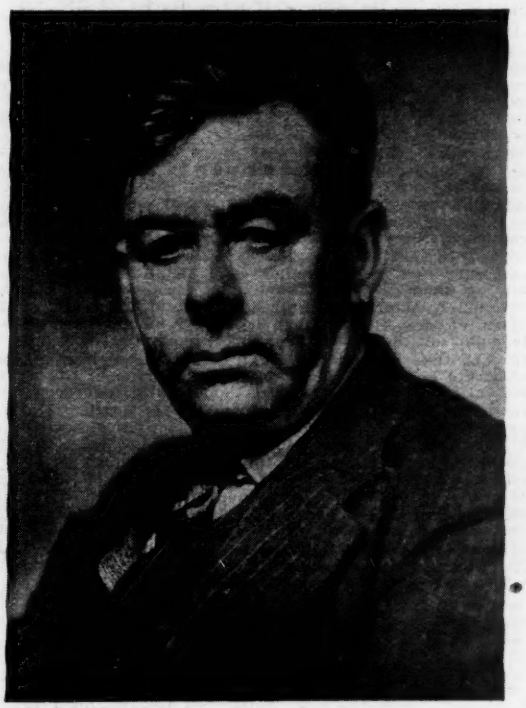
Being in no way parochial in outlook, his interests were not confined to his own hospital: he was appointed honorary radiologist to the Melbourne Dental Hospital in 1930 and honorary radiologist to the Victorian Eye and Ear Hospital in 1934, in both of which institutions he was to exhibit the same meticulous care and skill as was seen in his general radiology.

As a medical student he gave little indication of the brilliant career which lay ahead of him. Nevertheless, at that early stage he showed a wisdom beyond his years and exhibited in no uncertain fashion his ability to take the long-term view. Realizing the importance of a resident appointment at St. Vincent's, he elected to make certain of it by not sitting for his final examination with his contemporaries, but by waiting another year. For him there were to be no short cuts. It was this deliberate policy which was undoubtedly responsible for his success.

In 1923, whilst still registrar at St. Vincent's, he obtained his M.D. degree, and shortly afterwards left Australia for four years' study abroad. He passed the examination for the D.M.R. and E., Cambridge, in 1925. Dr. A. C. Barclay, one of the examiners at that time, wrote that he acquitted himself brilliantly and was the only man to whom a mark of distinction had been granted. Subsequently, he held the posts of senior assistant to the Radiological Department of the Manchester Royal Infirmary under Barclay and of assistant radiologist to the Royal Infirmary, Edinburgh, under Woodburn Morison. After leaving Edinburgh he studied in Germany, in Paris and in Vienna. In Berlin he was fortunate enough to work in the clinic of Professor Berg, who did so much to elaborate the precise technique for the examination of the stomach and duodenum and who had a great influence on the method of examination with the barium meal, in which John O'Sullivan was later to excel. In Vienna he first met Professor Schuller, and here began a friendship which was to ripen later at St. Vincent's, when the professor, in exile, sought refuge in Australia. Realizing to the full the value of continental radiology, he eventually subscribed to journals in French, German, Spanish and Italian. He returned to Australia in 1928 and was appointed honorary radiologist to St. Vincent's.

He was a daily attendant at Dr. Wright Smith's post-mortem demonstrations at the Royal Melbourne Hospital in the early 1930's, and was a constant visitor to the Pathology Department at St. Vincent's, where he was to be seen in consultation with Dr. Andrew Brenan, relentlessly pursuing his cases to their ultimate conclusion. This was a habit

acquired in Vienna, where he was greatly impressed by the post-mortem demonstrations of Erdheim. At this time, being interested in the implantation of radium needles for the treatment of cancer, he prepared himself for a surgical career by taking the first parts of the M.S. and of the F.R.A.C.S. All this was accomplished in the midst of a busy practice, the only time he could find to do his dissection being when his appointments were finished, in the late afternoon or at night. In following such a strenuous programme, he was striving to attain the standard of excellence of the radiologist described by him when he wrote: "I should like also here to stress that it is only by proper correlation between the radiological appearances, pathological changes and clinical findings that progress will be made. It is not sufficient for the radiologist to be satisfied with the production of a practically perfect negative. He is more than a photographer. What is required of him is a diagnosis of the existing pathological changes."[1]



Such was his devotion to duty that his last task for the day, often late at night, was to sign and dispatch reports on cases seen that day. He rose early and started reporting on films to the dictaphone in a special room at his house. It was usual for him to arrive at the Mercy Hospital at 6 p.m. on Saturday laden with the films on which he had been reporting all the afternoon. He read widely in his subject and was able to do so, as he often woke at two or three in the morning, and then read for several hours. He read with ease in five languages, and his wife tells of his reading the Bible in shorthand. His library reflected his taste as a collector and was filled with latest editions as well. He had a standing arrangement with a publishing house whereby any medical books likely to interest him were automatically sent for his perusal. Annotations on page after page of his books indicate that he remained to the last a student.

His many official positions bore witness to the high regard in which he was held by his colleagues. In 1936 he became a member of the Board of the Cancer Institute. He was Vice-President of the Radiological Section at the fifth session of the Australasian Medical Congress (British Medical Association) in Adelaide in 1937. In 1940 he was appointed specialist radiologist to the Royal Australian Air Force with the rank of wing commander, and in 1947 consulting radiologist to the Royal Australian Navy. In 1948 he was President of the Australian and New Zealand Association of Radiologists, and in 1950 he led the Australian delegation to the International Radiological Congress in London. In 1952 he took part in a symposium on the management of peptic ulcer with Sir Gordon Gordon-Taylor and Dr. Avery Jones at the eighth session of the Australasian Medical Congress (British Medical Association) in Melbourne. He was a member of the Executive and of the Scientific Committee of the Anti-Cancer Council of Victoria. He was a Fellow of The Royal Australasian College of Physicians, and he achieved the high distinction of Fellowship of the Faculty of Radiologists.

There was a human side to "Johnny". He was a devoted husband and father and was extremely happy in his home life, and our deepest sympathy is extended to his widow and only son. A connoisseur in the widest sense, a lover of books and music, he could still react to the youthful enthusiasm of his son and share his keenness for sailing and the sports car, remembering the *Itala* of his resident days, which was the envy of his colleagues. His collector's instinct was not limited to books alone, but extended to modern sports guns, ancient pistols and photographic equipment. At all times he had to have the right tool for the job. With prodigal extravagance he was always completely equipped for any emergency—if he went for a day's motoring, he loaded into his car not one, but several types of camera, guns, fishing rods, golf bags and breakdown gear. Although he might use none of them, they were there if the occasion should arise. Always ready with a laugh for the latest anecdote, he would nevertheless have us believe at times that civilization was rushing to its doom at ever-increasing speed; that, of course, he did not believe, and we took this to be a reflection from one of the many brilliant facets of an unusual personality. *Requiescat in pace.*

DR. KEITH HALLAM writes: John O'Sullivan was one of a small band of graduates from the Medical School of the University of Melbourne who went to Britain and Europe in the 1920's, and who by these experiences was able to be a force in the consolidation of radiology in Australia after the pioneering work of the early radiologists and physicists. They did much to advance and establish radiology on a clinical and physical basis and as an art.

John O'Sullivan prepared himself by long post-graduate study so that a Doctorate of Medicine at the University of Melbourne in 1923 and a Diploma in Medical Radiology and Electrology at Cambridge in 1925 followed his graduation in medicine and surgery in 1921. Subsequently he was awarded a Fellowship of The Royal Australasian College of Physicians and a Fellowship of the Faculty of Radiology of London. A knowledge of some European languages enabled him to have a first-hand contact with current medical literature. To this formidable acquisition of academic honours he added a meticulous study of the methods, equipment and physics of radiology, thus being always a complete master of the various phases of his chosen field in the practice of medicine.

He was intensely interested in corporate efforts to further radiology in Australasia, helping in the initiation of the Association of Radiologists in 1934, and sixteen years later in the inauguration of the College of Radiologists. For a long span he served on the Council of the Association, becoming its President in 1949.

As a teacher of radiology he was much sought after, and for many years was co-examiner in pathology for the diplomas of diagnostic and therapeutic radiology of the University of Melbourne. Other activities were membership of a cancer committee of the National Health and Medical Research Council, and of the Standing Committee for X Rays of this Council, where he did pioneering and yeomen service in the chest surveys for the control of tuberculosis. With Dr. (now Sir) Harry Wunderly and the late Dr. Eric Cooper, and Dr. Cecil Eddy (late director of the X-Ray and Radium Laboratory of the Commonwealth Health Department), he was the inspiring radiologist in a team which introduced X-ray microfilm surveys of the chests of members of the armed forces. This was begun in the early months of 1940, and continued with ever-increasing impetus throughout the war. In the post-war years he was a member of the Tuberculosis Committee of the Health Department of Victoria, which was an advisory body for the control of tuberculosis in the general population. He was also a member of the board of the Cancer Institute, now the Peter MacCallum Clinic, of which he was for a time the chairman.

This imposing array of his accomplishments as a leading figure in the medical life of the community must necessarily have stemmed from innate qualities of intellectuality, strong character, personality and drive. Added to these he had a quiet charm, a winsomeness and a genuine sympathy and kindliness which attracted people socially, and endeared him to his colleagues and his patients. Needless to say, he built up a huge practice, in which he was the supreme individualist, so that he did not absorb any partners,

[1] *Brit. J. Radiol.*, 1952, 5:103 (February).

resulting in overwork at his honorary duties and private practice. This led to his comparatively early death at the age of sixty. All patients, whether public or private, were treated with comprehensive understanding and gentleness, and many of his private patients were in fact honorary. In some cases, where he learned of hardship, the gift of something which would make the life of unfortunates easier was covertly bestowed.

He was always a staunch fighter for radiologists to save them from being overwhelmed by the rising tide of opinion over the years that investigational services should be fully salaried or "nationalized". To change the metaphor to his own expression, he was constantly appealing to all interested parties that radiologists "should not be thrown to the wolves" to satisfy the demands of free medical services to the public. In normal debate he expressed well-thought opinions in his quiet and gentle manner, but when he considered an injustice might be done, he blazed forth in loud and forceful protest. His classic expression at clinical meetings was a salutary one wherein he warned all and sundry not to be deluded by "fascination". Due to his meticulous and methodical nature, he had learned that the finding of one pathological change was not the end of the trail. Over-weening confidence and sense of fulfilment should not delude into overlooking signs in films of other, and perhaps more significant, lesions of a patient.

As a companion, either socially or travelling, he was genial, understanding and, most of all, helpful. One has many happy memories of journeys to other capitals with him and his charming wife, Serita. A cherished memory is our journey to Canberra in his La Salle car with its innumerable gadgets within and outside, dedicated to safety and to the coping with possible calamities. He loved equipment and gadgets of all kinds, so that there may be at hand efficient tools for the best possible end-result of any job, whether in his practice, his play, or the ordinary ways of life. Abstemious and religious, he could not abide over-indulgence or intolerance. I remember on one of our many meanderings round Canberra we were leaving the War Memorial when he remarked that it lacked any spiritual quality. He had felt that the atmosphere of this exquisite memorial was pagan.

He was a big man, but not physically strong. His stooped figure surmounted by a large head with its permanent shock of hair was striking in any company. His beaming smile and courtesy attracted, so that on meeting him one never had that chilling sense of vacuity which may happen on occasions. In controversy and battle when tempers were frayed, he held the view that his own code of behaviour should not be influenced and degraded by any underhand conduct of his adversaries.

In my long association with John O'Sullivan I felt he was a dedicated man who knew where he was going and what his aim was: perfection in all he attempted. Devotion was the keynote of his life: devotion to his work, his patients, his College of Radiologists, and most of all to his beloved wife and little John. With the passing of John O'Sullivan, a massive compendium of radiology has been lost to this world, but his teachings and his example in this field will live on.

DR. H. G. FURNELL writes: John O'Sullivan had become an institution at St. Vincent's Hospital. He was supreme in his field; the staff all felt that he was able to give the last word on any problem in his department; his skill and professional integrity were unquestioned. Not many men attain such a position, and his standing in his specialty was, of course, Australia-wide and not confined to the hospital. Such eminence is not easily obtained, nor lightly held. John O'Sullivan devoted his life to his specialty. The writer knew him as a student and as a new graduate, when already his future was clearly marked out in his own mind. After graduation, and a second year as registrar (the only senior resident position), which gave him an entry to the X-Ray Department, he went abroad. He spent several years in post-graduate study. At Edinburgh he worked under Woodburn Morison, becoming his assistant in the Radiology Department at the Royal Infirmary, at that time probably the most highly regarded in all Britain. Fortunately, he was financially able to devote these long years to unremunerative study, and fortunately for his hospital and for Australia, he made full use of his opportunities. On his return to Australia in 1928 he was appointed assistant to Dr. Herbert Hewlett, one of the pioneers of diagnostic radiology, who survived in honoured retirement until last year. John O'Sullivan succeeded Dr. Hewlett as radiologist to St. Vincent's in 1928. For years he combined diagnostic and therapeutic radiology. In the field of therapeutic radiology quite often he did not see eye to eye with his colleagues, but he always held firmly to his viewpoint, and disputation in no way lowered the high regard in which his colleagues held him. There are many more competent than I to speak of his position in Australian radiology. He held high office in the College of Radiologists of Australasia, being President, in 1949, of the Australian and New Zealand Association of Radiologists before the College was founded.

Apart from his work, John O'Sullivan was a home lover. He was fortunate in having a very happy home. To his devoted wife and son we tender our most sincere sympathy, and we pay our tribute to a man who was an honour to the profession he served with such devotion.

GEORGE HAMLYN HARRIS.

WE are indebted to Dr. W. H. Hill for the following appreciation of Dr. George Hamlyn Harris.

Dr. George Hamlyn Harris died suddenly at his home in Launceston, Tasmania, on December 25, 1957. He was 64 years of age. His early life was spent in New South Wales. He was educated at King's College and began his medical course in 1913 at the University of Sydney. His studies were interrupted by the first World War. He served with his three brothers in the 1st Light Horse Regiment in Palestine and Egypt and was twice wounded. After his military service he resumed his study of medicine, and after graduation and two years' hospital work he came to Tasmania and practised for four years on the north-west coast before coming to Launceston. He built up a large practice in the city which he carried on successfully for 25 years until laid aside by illness. His retirement was only temporary and his colleagues were delighted when he was able to undertake even part-time work again. He was particularly interested in obstetrics, and his work at the Queen Victoria Hospital will be long remembered. He was chairman of the Board of Management for six years. A wing of that hospital has been named after him to commemorate his work. He was a keen member of the British Medical Association and was honorary treasurer of the Northern Subdivision at the time of his death. He took active part in church and community activities, particularly in the Returned Servicemen's League, in which he was president of the local sub-branch. Perhaps he will be remembered best by his Christian devotion to his fellow men. He was beloved by his patients and esteemed by his colleagues, for he possessed that innate courtesy and kindliness which made him a friend of all and which inspired a warm affection in all those who worked with him. The world is a poorer place for his passing, and with his widow and family we mourn his loss.

MABEL IRENE COUSAN BRAY.

WE are indebted to Dr. Marjorie Tunley for the following appreciation of Dr. Mabel Irene Bray.

The death of Irene Bray on May 10 plunged Cassilis and district into mourning. She was the perfect family doctor, who not only dealt most ably with her patients' ailments, but was also their friend and trusted counsellor. She graduated in arts before studying medicine, and took an active part in University of Sydney and Women's College life. After graduation she was a resident medical officer at Royal Prince Alfred Hospital, and on completion of her term there she commenced practice in Cassilis, where she lived and worked for thirty-four years. By day and night, in all weathers and under all circumstances, she was at the service of those who needed and trusted her. She had a high standard of medical practice, and was a member of the Western Medical Association from the time of her arrival in Cassilis. She was recently elected to membership of the College of General Practitioners, and was keenly looking forward to their next week-end meeting at Manly. On May 7 she suffered a stroke, and died three days later without recovering consciousness. She leaves a host of friends and patients who will always remember her with affection and gratitude.

She was a very feminine person, and had a great charm of manner and sense of humour which endeared her to all. Keenly interested in history, music and art, her two trips overseas gave her endless pleasant memories. Her kind and sympathetic nature was allied to a great strength of purpose and courage. Only those who have practised their pro-

fession alone, many miles from another fellow practitioner, can realize how often these attributes were called upon to sustain her over the long years of service. Above all, she had high and rare qualities of heart and mind and spirit which will forever be cherished by those privileged to call her friend. We shall not see her like again.

Correspondence.

STAFFING OF BASE HOSPITALS.

SIR: The letter of Dr. J. G. Glasson in the Journal of April 26 and the reply of Dr. Hugh Hunter in the Journal of May 10 are worthy of comment. If my recollection is correct, the B.M.A. circular of March 17 read after "base hospitals should be staffed by classified staffs", "as qualified or trained people become available" Dr. Glasson would seem to see the problem only from his own side. Surely the patient must come first; and we, as a profession, should see that a person trained and qualified for the particular job does do that job whenever possible. No one can claim that a doctor is fully trained on graduation. Alas we are not fully trained when fully qualified. I have seen M's.R.C.O.G. who have never done a hysterectomy on their own, and I have seen country M.B's. and B's.S. who could put the M.R.C.O.G. to shame. The B.M.A. policy is to be wholly commended, and it would seem that the idea of honorary and associate honorary, as recently instituted in the new outer metropolitan hospitals, is worthy of an extended trial.

Yours, etc.,

19 Gerrale Street, Cronulla, New South Wales. A. M. MACINTOSH.

May 14, 1958.

ANÆSTHESIA IN HEART DISEASE.

SIR: There is one really serious aspect of Mr. Susman's criticism (M. J. AUSTRALIA, May 3, 1958) of my article on the above subject, and that is his persistent advocacy of hypnotic anæsthesia. In this regard I am privileged to quote a personal communication from Dr. Ainslie Meares, a recognized authority on hypnosis. This runs as follows:

> As regards the use of hypnosis for anæsthesia, in my opinion there are very real psychological hazards. When hypnosis is induced for any purpose, there is always some degree of interference with the effectiveness of the repressive mechanism; in fact this is the basis of the use of hypnosis as a means of uncovering suppressed and repressed psychological conflicts. When hypnosis is induced for purposes of anæsthesia, there tends to be a loss of effective psychological repression. The result is that the patient may experience a sudden surge into consciousness of extremely traumatic repressed material. The content of this material may include such ideas as thoughts of incest or homosexuality. With a patient who has never suspected that he harbours such ideas, when these thoughts suddenly surge into consciousness, he is stricken with an acute anxiety reaction which may be very difficult to control if the hypnotist is not really experienced in dealing with anxiety and abreaction in the hypnotized patient. The situation must be met by increasing the depth of hypnosis, rather than by waking the patient, which would seem to be the natural thing to do when things are getting out of hand. It would seem to me that the anæsthetist who has had no psychiatric training in hypnosis might find it difficult to do this.
>
> The anæsthetist hypnotizes the patient, and gives him the suggestion of anæsthesia. The patient is then tested for anæsthesia by grasping the skin in artery forceps. It would seem simple enough, if the suggestions have failed, and the patient still flinches, that the anæsthetist could then proceed with pharmacological anæsthesia. This would fulfil the anæsthetic requirements of the situation, but it would seem to do nothing to resolve the patient's anxiety as the result of the unfulfilled hypnotic suggestion. I simply do not know if subsequent anæsthesia has any effect in these circumstances, but I think it likely that it would not allay the anxiety, as in ordinary clinical hypnosis, such anxiety is often manifested in psychosomatic equivalents.
>
> There are further psychological hazards. There is very good evidence that the pre-psychotic schizophrenic may be precipitated into frank psychosis by hypnosis. It would seem that the pre-psychotic schizophrenic harbours vague, ill-formed ideas of influence. Hypnosis in such persons may crystallize, as it were, these vague feelings into definite delusional thinking. It would seem to me unlikely that the anæsthetist untrained in psychiatry would be able to recognize such patients. In this respect, I am referring to persons who show no obvious outward signs of a pre-psychotic condition.
>
> I think the anæsthetist who became known as one using hypnosis would be involved in another psychological hazard. This comes from perverse motivation from the patient. I myself find that I am frequently consulted by patients who desire to be treated by hypnosis for some nervous disorder. But from psychological examination of the patient, it soon becomes clear that the patient is not so much seeking treatment of his nervous condition, but is rather seeking hypnosis for itself. This is seen in masochistic women who have a yearning for the experience of being overpowered, which they expect to find in hypnosis. Other women, of masculine-aggressive personality, seek hypnosis in the firm belief that they will not be hypnotized, and will so be able to satisfy their latent aggressive tendencies in proving that they cannot be overpowered by any man. Other patients, of both sexes, often seek hypnosis for the close emotional relationship which it will give them with the hypnotist. They unconsciously interpret this at an erotic level: the male patient on a homosexual basis, and the female patient on a heterosexual basis. The pre-psychotic schizophrenic often produces some rationalization to seek hypnosis, when in actual fact he is drawn to it by his ill-defined feelings of influence. The anæsthetist practising hypnosis might well find it hard to evaluate the motivation in these patients because, unlike the psychiatrist, he is not really in a position to make a psychological examination of the patient prior to hypnosis.
>
> There is a further psychological hazard. It seems that there are some patients who, when they are hypnotized, are not anæsthetic, but at the same time they seem to have some extraordinary capacity to suffer pain without complaint. I am not sure of the mechanism of this condition, but it at least concerns the intense rapport between patient and hypnotist, and probably has some relationship with latent guilt in the patient, so that the patient is really expiating his guilt by suffering. There is good evidence that this mechanism occurs when hypnosis is used in obstetrics.
>
> There is another hazard which is really a technical one, rather than a psychological one. I am sure that the old concept of levels of hypnosis is invalid. This idea pre-supposed a certain constancy, so that a person could be considered to remain at a certain level of hypnosis. In actual fact, with many patients the depth of hypnosis may be a constantly varying factor; not from five minutes to the next five minutes, but actually from moment to moment. I think that these variations are very largely a function of the imperfect repression, and the consequent surge of repressed material towards the threshold of consciousness.

It will be observed that Dr. Meares refers above specifically to the anæsthetist, but obviously this term must include any amateur exponent of hypnotic therapy.

In conclusion, I thank Mr. Susman for his generous acknowledgement of the help given by me and other anæsthetists in the past.

Yours, etc.,

143 Macquarie Street, Sydney, S. V. MARSHALL.

May 20, 1958.

[NOTE: This correspondence is now closed.—EDITOR.]

NATIONAL HEALTH SERVICE.

SIR: Various rumours come through conversational circles in the profession about the possibility (or threat?) of a compulsory medical service of national type for Australia. Are we doing anything practical to construct a defence against insurance experts or ambitious politicians when the time comes? For those of us who are content to take it, I proffer a bittie of information which has reached me from Scotland. In that excellent country there is a town called

Callander. It is a small town in the rather remote fastnesses of the trossachs. The local bosses of the National Medical Service advertised a post for a general practitioner recently. For this one post there were seventy-eight applicants. If ever it comes to us, we cannot say we have not been warned.

Yours, etc.,

JOHN A. McCLUSKIE.

133 Wigram Road,
Glebe, Sydney,
May 15, 1958.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Week-End Course in Gastro-Enterology.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that a week-end course in gastro-enterology for general practitioners will be held in the Scot Skirving Lecture Theatre, Royal Prince Alfred Hospital, Camperdown, on Saturday and Sunday, June 21 and 22, 1958, under the supervision of Dr. Stanley Goulston. The chairman will be Dr. A. W. Morrow. The theme of the course this year is "Disorders of the Colon". The programme is as follows:

Saturday, June 21: 9.30 a.m., "Normal and Disordered Function in the Colon and Rectum", Dr. D. Piper; 10 a.m., "The Clinical Investigation of Large Bowel Disorders"—(*a*) stool examination, Dr. Edgar Thomson, (*b*) radiology, Dr. Alan Colwell, (*c*) endoscopy, Dr. John Spence; 11.30 a.m., "The Significance and Investigation of Blood in the Stools", Dr. John Sands; 12 noon, discussion; 12.15 p.m., "The Causes and Management of Large Bowel Diarrhœa", Dr. Bruce Hall; 12.45 p.m., discussion.

Sunday, June 22: 9.30 a.m., "Colonic Motor and Secretory Disturbances in 'Spastic Colon'", Dr. Brian Billington; 10 a.m., discussion; 10.15 a.m., "The Management of Constipation (Including Megacolon)", Dr. W. B. Hennessy; 11.30 a.m., "Diverticulosis and its Complications", Mr. S. H. Lovell; 12 noon, discussion; 12.15 p.m., "Polyposis of the Colon", Professor John Loewenthal; 12.45 p.m., discussion; 1 p.m., lunch by courtesy of the Board of Directors, Royal Prince Alfred Hospital; 2 p.m., symposium on "Ulcerative Colitis"—(*a*) classification, clinical aspects and complications, Dr. Stanley Goulston, (*b*) proctosigmoiditis, Dr. R. Boden, (*c*) ileo-colitis, Dr. D. Church, (*d*) management, Dr. David Maddison, (*e*) management, Dr. Stanley Goulston, (*f*) management, Mr. N. R. Wyndham; 3.30 p.m., discussion; 4.30 p.m., question time—panel, Dr. A. W. Morrow, Dr. Bruce Hall, Dr. Brian Billington, Dr. D. Piper, Dr. Stanley Goulston.

The fee for attendance is £3 3s., and those wishing to attend are requested to make written application, enclosing remittance, to the Course Secretary, The Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephone: BU 4497-8.

THE MELBOURNE MEDICAL POST-GRADUATE COMMITTEE.

PROGRAMME FOR JUNE, 1958.

Courses for Higher Qualifications.

Course in Pathology.—A course in pathology suitable for candidates for primary F.R.A.C.S. will commence on Monday, June 30, at the Pathology Department, University of Melbourne. A series of twelve lectures will be given on Monday and Wednesday afternoons from 1.30 p.m. to 2.30 p.m. The fee is £7 7s., and enrolments should be made by June 16 on the Committee's special form. Anatomy lectures will begin from June 30 at 2.45 p.m.

Course in Psychiatry.—A course in psychiatry suitable for candidates for D.P.M. will commence on Thursday, June 12. The Australasian Association of Psychiatrists will conduct a series of sixteen lectures in psychiatry on Thursdays, at 8.15 p.m., in the Small Lecture Theatre, Royal Melbourne Hospital. The programme will include discussions on the psychotherapy of everyday practice, the clinical applications of psychoanalysis, psychotherapy in children and parents, creative activity as a psychotherapeutic agent, schizophrenia, organic approaches in psychiatry, the chronic insane patient, and the psychiatrist in court. The fee for this course is £10 10s., and enrolments should be made with the Post-Graduate Committee by May 29.

Each Wednesday during the currency of this course the honorary staff of the Psychiatric Unit of the Royal Melbourne Hospital will conduct a post-graduate teaching session from 2.45 to 4.30 p.m., to which members of the class will be invited.

Course in Medicine.

The full-time course in medicine at Prince Henry's Hospital, suitable for candidates for higher qualifications, which commenced on May 26, will continue throughout June.

Country Courses.

Wangaratta.—On Saturday, June 7, at the Base Hospital, Wangaratta: 2.30 p.m., "Management of Hypertension", Dr. Austin Doyle; 3.45 p.m., "Hæmaturia", Mr. J. B. Somerset; 4.45 p.m., "Habitual Abortion", Mr. W. J. Rawlings.

The fee for the course is 15s. per lecture, but those who have paid an annual subscription to the Committee may attend without further charge. The local secretary is Dr. M. Rohan, 10 Docker Street, Wangaratta. Telephone: Wangaratta 602.

Flinders Naval Depot.

On Wednesday, June 11, at 2.30 p.m., Dr. T. H. Hurley will talk on "Adverse Reactions to Drug Therapy". This lecture will be given by arrangement with the Royal Australian Navy.

Course in Neuropathology.

Those interested in a short course in neuropathology are asked to contact the Post-Graduate Committee.

Information.

The address of the Melbourne Medical Post-Graduate Committee is 394 Albert Street, East Melbourne. Telephone: FB 2547.

Medical Societies.

SOCIETY FOR CLINICAL SCIENCE.

THE next meeting of the Society for Clinical Science will be held at the Royal Alexandra Hospital for Children, Camperdown, in the Doreen Dew Lecture Theatre, at 7.15 p.m. on Thursday, June 5, 1958. Dr. C. W. G. Lee and Miss A. E. Stobo will speak on "Some Aspects of Galactose Intolerance". Dr. K. Mattocks will be in the chair. The laboratories will be open to inspection. The meeting is open to all interested.

Direction notices will be found at the front entrance. At night visitors' cars may be parked within the grounds at the front of the hospital.

THE ENDOCRINE SOCIETY OF AUSTRALASIA.

A MEETING relating to the proposal to form an Endocrine Society of Australasia will be held in the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney, on Monday, June 2, 1958, at 2.15 p.m. Its purpose will be to receive the report of an interim committee which has been exploring the above-mentioned proposal to proceed, if thought desirable, to the formation of such a society and to adopt a constitution. Those interested are invited to attend. The acting secretary of the interim committee is Dr. Bryan Hudson. The other members of the committee are Dr. Keith Harrison, Dr. Basil Hetzel, Dr. Ewen Downie and Dr. Pincus Taft.

A scientific session will be held on Tuesday, June 3, 1958, in the Robert H. Todd Assembly Hall, and all interested are invited to attend. The programme is as follows: 9.30 a.m., "Studies of Pregnanetriol and Related Steroids", Dr. R. I. Cox, Department of Veterinary Physiology, University of Sydney; 10 a.m., "Neurohypophyseal Function", Dr. Peter Hall, Sydney Hospital; 11 a.m., "The Estimation of Urinary 17-Ketosteroids: An Appraisal of Current Methods", Miss June Sheath, Diabetic and Metabolic Unit, Alfred Hospital, Melbourne; 11.30 a.m., "Amenorrhœa and Virilism in Liver Disease", Dr. W. Hamilton Smith, St. Vincent's Hospital,

Melbourne; 2 p.m., "Thyroid Hormone Flow in Euthyroid Subjects with High Iodine Uptake Rates", Dr. I. D. Thomas, Dr. T. H. Oddie and Dr. F. F. Rundle, Thyroid Investigation Clinic, Institute of Medical Research, Royal North Shore Hospital, Sydney; 2.30 p.m., "Observations on Body Hair", Dr. H. P. Taft and Dr. R. Melick, Royal Melbourne Hospital; 3.30 p.m., "Some Properties of an Oxytocic Substance Found in Blood and Hypothalamic Extracts", Dr. R. W. Hawker, Department of Physiology, University of Queensland; 4 p.m., "Studies on the Metabolic Effects of Salicylate in Man", Dr. Basil S. Hetzel, Department of Medicine, University of Adelaide.

Notice.

NAPT COMMONWEALTH CHEST CONFERENCE.

THE NAPT Commonwealth Chest Conference, incorporating the annual conference of the British Tuberculosis Association, will be held at the Royal Festival Hall, London, from July 1 to 4, 1958. The provisional programme includes the following items:

Tuesday, July 1: 10.30 a.m., discussion at B.M.A. House on "Tuberculosis Care Work in the Commonwealth" for Commonwealth delegates; 6.30 p.m., inaugural meeting and reception—official opening by H.R.H. the Duchess of Kent.

Wednesday, July 2: "The World Anti-Tuberculosis Campaign: Is it Succeeding?"; speakers will include Sir Harry Wunderly (Australia), Dr. B. A. Dormer (Union of South Africa), Sir Kenneth Cowan, Dr. Johannes Holm (WHO), Dr. J. H. F. Jayasuriya (Ceylon) and Dr. R. T. Neubauer (Yugoslavia). The titles of other meetings include: "Danger of T.B. Infection to Personnel in Pathological Laboratories", chairman, Dr. G. S. Wilson; "Towards the Elimination of Tuberculosis in Great Britain", chairman, Dr. C. H. C. Toussaint; "Tuberculosis and Leprosy: Resemblances and Divergences in Diagnosis and Treatment", chairman, Dr. F. A. H. Simmonds; "Respiratory Disease: A Malady of Environment", chairman, Dr. J. Greenwood Wilson; "The Management of the Heart in Chronic Respiratory Disease", chairman, Dr. Robert Platt.

Thursday, July 3: "Tuberculosis Medical and Social Services in the British Commonwealth", chairman, Mr. Anthony Greenwood, M.P.; "The Heart Patient in Everyday Life", chairman, Dr. K. Shirley Smith; "Fungus Disease of the Lung. Pneumoconiosis. Immunity Reactions in Tuberculosis and Sarcoidosis", chairman, Dr. W. E. Sneli; "The Patient's personality", chairman, Dr. R. R. Trail; "NAPT Section Meeting—BCG Vaccination", chairman, Dr. G. Lissant Cox.

Friday, July 4: "The Family and the Patient with Chest Disease: How Best Can We Help Them to Help Themselves?", chairman, Sir Robert A. Young; "Thoracic Surgery in Pulmonary Tuberculosis", chairman, Sir Clement Price Thomas; "The Problem of Lung Cancer", chairman, Dr. N. Lloyd Rusby.

The full membership fee is £5 5s., or 15s. per session. The conference is open to all interested in preventive medicine, including the medical and nursing professions, research workers, health administrators and others. Further information may be obtained from the Secretary-General, National Association for the Prevention of Tuberculosis, Tavistock House North, Tavistock Square, London, W.C.1.

BRITISH MEDICAL ASSOCIATION, NEW SOUTH WALES BRANCH.

Special Group of Neurology and Psychiatry.

IN the monthly bulletin of the New South Wales Branch of the British Medical Association, dated May 19, 1958, the date of the meeting of the Special Group of Neurology and Psychiatry on "Leucotomy: The Present and the Future" was incorrectly advertised. This meeting will be held at Sydney Hospital at 8 p.m. on Thursday, June 5, 1958.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED MAY 10, 1958.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.[2]	Australia.[3]
Acute Rheumatism	3	2(2)	..	..	1	..	..	..	6
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	5	..	5
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	1	..	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	1	..	..	..	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	3(1)	13(12)	2(2)	..	..	..	..	..	18
Diphtheria	1	2(2)	1	..	1(1)	..	..	..	5
Dysentery (Bacillary)	..	32(2)	1(1)	1(1)	1(1)	..	2	..	37
Encephalitis	..	1(1)	..	..	..	..	..	..	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	1	..	..	1	..	2	..	..	4
Infective Hepatitis	46(27)	15(5)	3(1)	3(2)	3(1)	1(1)	..	..	71
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	4	..	..	..	..	..	4
Malaria	..	..	1[4]	..	..	..	..	..	1
Meningococcal Infection	1(1)	1(1)	..	..	..	..	..	..	2
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	2(1)	..	..	..	..	..	..	..	2
Puerperal Fever	..	..	1	..	..	..	..	..	1
Rubella	..	11(9)	..	1	21(16)	..	..	..	33
Salmonella Infection	..	..	..	..	1(1)	..	..	..	1
Scarlet Fever	17(10)	29(21)	5	..	5(4)	2	..	..	58
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	1	1(1)	..	..	..	..	2
Trachoma	..	..	..	..	10(4)	..	2	..	12
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	24(20)	25(16)	32(11)	10(8)	5(4)	4(1)	..	..	100
Typhoid Fever	..	2(2)	..	1(1)	..	..	..	..	3
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from Australian Capital Territory.
[4] Infection contracted outside Australia.

Medical Prizes.

THE DARCY COWAN PRIZE.

THE Darcy Cowan Prize has been founded by the Australian Laennec Society (the medical section of the National Association for the Prevention of Tuberculosis in Australia) to honour the work of Sir Darcy Cowan, a prominent Adelaide physician, in the field of tuberculosis in Australia. It is expected that this prize will be offered each year, and that it will be awarded first at the next national meeting of N.A.P.T.A. in Melbourne in February, 1959.

The particulars of this prize are as follows: (i) Its value is £50. (ii) It is to be awarded for the best essay on any subject relating to respiratory disease. It should include original work and observations. (iii) It is open to all medical graduates of Australian universities of not more than five years' standing or thirty years of age.

Essays (three copies of double-spaced typing on foolscap) must be in the hands of the National Secretary by November 30, 1958. They should be addressed to Dr. R. Munro Ford, 163 North Terrace, Adelaide, South Australia. Further particulars concerning this prize may be obtained from Dr. Ford.

AUSTRALIAN SOCIETY OF ANÆSTHETISTS' PRIZE FOR ANÆSTHETIST REGISTRARS.

THE Australian Society of Anæsthetists offers an annual prize of £25 for the best original paper submitted by an Australian anæsthetic registrar.

Award for 1957.

The prize for 1957 was awarded to Dr. Brian Crawshaw, of New South Wales.

Conditions of Entry for 1958.

1. The Australian Society of Anæsthetists awards an annual prize of £25 for the best original paper submitted by an Australian anæsthetist holding the grading of senior registrar or under. It is not necessary that he or she be a member of the Society.

2. The subject of the paper is optional, but must be related to anæsthesia. It is desirable that papers submitted show evidence of personal work, but papers consisting of surveys of the literature are eligible for consideration. The paper must not have been previously read at any meeting or published in any journal.

3. The winner of the prize will be invited to read his paper at the annual general meeting of the Australian Society of Anæsthetists. The Society will give every assistance in obtaining publication of the paper in a recognized medical journal.

4. Papers for adjudication should reach the honorary secretary, Dr. Patricia Wilson, 132 Grey Street, East Melbourne, Victoria, not later than December 1, 1958.

Nominations and Elections.

THE undermentioned has applied for election as a member of the Victorian Branch of the British Medical Association:

Russell, Yvonne Kerr, M.B., B.S., 1949 (Univ. London), D.A. (Univ. London), 1 Myross Avenue, Ascot Vale West, Victoria.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association: Ham, John Mackenzie, M.B., B.S., 1957 (Univ. Sydney); Pegum, Brian Francis, M.B., B.S., 1957 (Univ. Sydney); Bench, Graham John, M.B., B.S., 1958 (Univ. Sydney); Panettiere, Noel Daniele, M.B., B.S., 1958 (Univ. Melbourne); Jones, Louise Elizabeth, M.B., B.S., 1958 (Univ. Sydney); Cutler, Geoffrey Abbott, M.B., B.S., 1952 (Univ. Sydney); Fisher, Harold Anthony, M.B., B.S., 1956 (Univ. Sydney); Lacey, Richard Grove, M.B., B.S., 1954 (Univ. London); Leitch, Austen Harding, M.B., B.Ch., 1950 (Univ. Witwatersrand); Dichne, David, M.D., 1927 (Univ. Leipzig); Harmos, Gyula Arpad Ferenc, M.D., 1937 (Univ. Pecs); Lucas, Joseph Anthony, M.D., 1941 (Univ. Bratislava); Burrow, Marea Graham, M.B., B.S., 1957 (Univ. Sydney).

Deaths.

THE following deaths have been announced:

BRAY.—Mabel Irene Cousan Bray, on May 10, 1958, at Cassilis, New South Wales.

GLISSAN.—Denis Joseph Glissan, on May 19, 1958, at the Repatriation General Hospital, Concord, New South Wales.

Diary for the Month.

JUNE 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JUNE 4.—Western Australian Branch, B.M.A.: Branch Council.
JUNE 5.—South Australian Branch, B.M.A.: Branch Council Meeting.
JUNE 6.—Queensland Branch, B.M.A.: General Meeting.
JUNE 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 12.—New South Wales Branch, B.M.A.: Public Relations Committee.
JUNE 13.—Tasmanian Branch, B.M.A.: Branch Council.
JUNE 13.—Queensland Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales. Anti-Tuberculosis Association of New South Wales.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this Journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in Australia can become subscribers to the Journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £5 *per annum* within Australia and the British Commonwealth of Nations, and £6 *per annum* within America and foreign countries, payable in advance.